INHERITANCE OF VENTRAL SPOTTING IN MICE

J. M. MURRAY AND C. V. GREEN
Roscoe B. Jackson Memorial Laboratory,
Bar Harbor, Maine

Received February 3, 1933

The two best known types of white spotting in mice are "black-eyed white" and "piebald". The former, *Ww*, is dominant (lethal in a homozygous condition) while the latter, *ss*, is recessive to self. A possible third distinct form, "all white" (*whwh*) has been mentioned by KEELER (1931). In addition, a number of other types, at least some of which are the result of modifying genes on basic piebald, have been noted in the literature.

DUNN (1920, 1925) described two modifications of the piebald pattern. In one, which he designated as "white face spotting", the white areas were restricted to the face; and in the other, "belt", to a band around the body. By inbreeding, he succeeded in fixing the white face pattern but was less successful with belt.

LITTLE (1917, 1926) reported a form of spotting, "blaze", in which the animals showed a small, white forehead spot. Besides the main gene responsible for this condition, he concluded that there were several modifying genes influencing the amount of white.

The same author (1924) observed two localized forms of spotting, "white tail tip" and an unpigmented band around the tail, "white on tail". In the race in which these types were observed, he reported that occasionally the tail spotting was transferred to the belly, such animals behaving in inheritance like phenotypically tail-spotted mice.

Ventral spotting appears to be of rather frequent occurrence in mice. DUNN (1920) found its appearance dependent on a modifying gene in conjunction with piebald in the heterozygous condition (*Ss*). ALLEN (1914) considered a mid-ventral patch generally to be the first unpigmented area developing in mammals.

In one inbred strain in our laboratory more than 50 percent of all individuals show a white ventral patch. The mice of this strain (C-57 Brown) are intense brown non-agouti (*DDbbaa*) in color and directly descended, by brother-to-sister matings with some backcrosses, from two control animals of one of Dr. C. C. LITTLE's previous experiments. The mice reported on in this paper comprise undepleted litters of the thirteenth to the twenty-eighth filial generations, inclusive.

The patches vary in size from 0 to approximately 10 percent of the ventral surface. In the few mice of the highest grades the unpigmented area sometimes extends up onto the side of the animal. All determinations

of grade were made by the senior author, thus reducing the personal error incident to more than one observer, while the junior author is responsible for the computations and analysis. Some animals exhibited "white on tail" or "white tail tip" with or without ventral spotting. It seems probable, however, that in the strain of browns the tail markings and ventral spotting are not conditioned by the same factors. The C-57 Blacks in which LITTLE described tail spotting still produce a majority with such markings while belly spotting is now almost non-existent. The C-57 Browns, with a common origin, show both tail and ventral spotting. Moreover, satisfactory records as to the degree of tail spotting manifested by individual mice were not available so it was deemed advisable to consider only ventral white.

The distribution of the 2166 mice of undepleted litters by grade of ventral spotting is shown in table 1.

TABLE 1

Distribution of offspring in brown stock grade of ventral spotting.

	0	1	2	3	4	5	6	7	8	9	10	TOTAL
Sexes combined	984	658	260	92	80	46	13	9	15	4	5	2166
Percent	45.4	30.4	12.0	4.2	3.7	2.1	0.6	0.4	0.7	0.2	0.2	
Males	420	340	154	60	55	25	8	3	10	3	3	1081
Percent	38.6	31.5	14.2	5.6	5.1	2.3	0.7	0.3	0.9	0.3	0.3	
Females	563	319	106	32	25	21	5	6	5	1	2	1085
Percent	51.9	29.4	9.8	2.9	2.3	1.9	0.5	0.6	0.5	0.1	0.2	

From this table it is evident that not only are more males than females spotted but also that the grade of the former tends to be higher.

Table 2 gives distributions and mean grades of offspring from matings differing in degree of ventral spotting.

TABLE 2

Distribution of offspring in different types of matings.

MATING		GRADE OF VENTRAL SPOTTING											MEAN
		0	1	2	3	4	5	6	7	8	9	10	
♀ Self × ♂ self	♂	130	82	36	7	5	2	1	1	1			.85
	♀	173	82	17	5	4	4		1	1			.63
♀ Grade 1 ×	♂	38	37	15	11	10	5	2		1		1	1.63
♂ Grade 1	♀	65	30	14	2	2	4	1	3			1	1.06
♀ Grade 2–7 ×	♂	22	23	9	7	6	6		2	5		1	2.22
♂ Grade 2–10	♀	27	26	6	6	5	6	1				1	1.56

It seems clear that a distinct correlation is present between grades of spotting in parents and offspring. This correlation, however, is imperfect since self parents produce offspring with a high grade of ventral spotting while spotted parents give birth to self animals.

The great majority of mice in the strain of browns are of three lines, A, C and CD. If the degree of ventral spotting is influenced by genetic factors, it might be expected that the lines would exhibit differences in the presence and extent of unpigmented areas. The distribution of ventral spotting in the three lines is listed in table 3.

TABLE 3

Distribution of ventral spotting in the three chief lines.

		GRADE OF VENTRAL SPOTTING											
		0	1	2	3	4	5	6	7	8	9	10	MEAN
Line A	♂	32	22	15	2	6	2			1			1.25
	♀	40	27	10	7	2	2		1				1.04
Line C	♂	122	102	37	8	9	6	3	1				1.01
	♀	178	96	26	2	9	5		3	2			.79
Line CD	♂	251	181	92	47	30	15	4	2	8	3	3	1.35
	♀	318	181	65	26	13	13	5	2	2	1	2	.95

The anticipated differentiation in the lines has apparently taken place since the mean grade is significantly lower, at least for the males, in Line C than in Line CD. Line A appears to be similar to CD although the numbers are few.

Since tables 2 and 3 make it seem probable that the presence and degree of ventral spotting are in part at least determined by genetic factors, parent-offspring correlation tables were constructed in order to show more exactly the extent to which variation in parental grade influenced variation in the grade of the offspring. The correlation between mid-parental grade and grade of offspring was $+0.258 \pm .014$. The square of this figure would indicate that slightly over six percent of the variation among the offspring was determined by variation among the parents.

The question which next suggests itself is the relative degree to which sire and dam influence spotting in the offspring. These correlations are given below:

Grade of spotting of sire—Grade of offspring $r = +0.269 \pm .013$

Grade of spotting of dam—Grade of offspring $r = +0.145 \pm .014$

These figures show that the correlation is positive and significant in both cases, but markedly higher with the sire than with the dam. A truer picture of the relative effects of the two parents may be given by the use of partial

correlations. This discloses that the correlation between sire and offspring with dam held constant is +0.243; that between dam and offspring with sire held constant is +0.086. The difference in the apparent influence of the two parents is thus further augmented.

The coefficients of correlation above have not taken into consideration the sex of the offspring. This factor is dealt with in the figures following:

	r	r (partial correlation)
Grade of spotting of sire —Grade of male offspring	+0.300±.019	+0.268 (Dam held constant)
Grade of spotting of dam —Grade of male offspring	+0.168±.020	+0.093 (Sire held constant)
Grade of spotting of sire —Grade of female offspring	+0.226±.019	+0.208 (Dam held constant)
Grade of spotting of dam —Grade of female offspring	+0.118±.020	+0.077 (Sire held constant)

Both sire and dam show slightly higher correlations with male than with female offspring although the differences probably lack significance.

It appears from the available evidence that heredity accounts for only a small portion of the variation in degree of ventral spotting in our strain of mice. The possibility that ventral spotting is due to either a single dominant or recessive gene is at once precluded. If dominant, self animals could not throw spotted offspring while if it were recessive, self young could not be produced by spotted parents. Of course if normal overlaps were postulated, these objections might be vitiated. That our ventrally spotted mice, like DUNN'S, are heterozygous piebalds is likewise precluded, since matings involving such parents do not produce the requisite piebald young. A number of genes may be involved although it might be expected that practical homozygosity would have been attained after 13 generations of inbreeding. The reason for the greater influence of the male in determining the grade of spotting of the offspring is not at all clear.

Since heredity—as measured by the square of the parent-offspring coefficient of correlation—accounts for but little more than six percent of the variance, non-genetic factors must play the major part in determining the degree of ventral spotting. The data permit an analysis of several such possibilities.

WRIGHT (1926) found that the age of the dam was of more importance than heredity in determining the grade of spotting in an inbred race of guinea pigs. In our material, there was present a correlation of +0.105±.014 between age of dam (in almost all cases the sire was of the same age) and grade of ventral spotting. Although significant, this is very low and would indicate that age accounts for about one percent of the variation in amount

of white in the offspring. Birth rank (position of litter in the series) likewise proves of slight importance as a factor with r equalling +0.068. In this case only males, both parents of which were self, were included. Since birth rank is largely a function of age it was to be expected that similar results would be encountered.

Still another possibility is that season of the year is a factor in influencing ventral spotting. Table 4, comprising offspring of self×self matings, affords scant evidence of this.

TABLE 4

Time of year and ventral spotting.

SEASON OF BIRTH		GRADE OF VENTRAL SPOTTING									TOTAL	MEAN
		0	1	2	3	4	5	6	7	8		
Dec. Jan. Feb.	♂	47	19	6	1	1	2				76	.63
	♀	45	20	5	1		2				73	.59
Mar. Apr. May	♂	29	27	8	2	1				1	68	.90
	♀	40	27	3		2	1			1	74	.73
June July Aug.	♂	17	8	4	2						31	.71
	♀	24	15	3	1	1	1				45	.73
Sept. Oct. Nov.	♂	37	28	18	2	3		1	1		90	1.06
	♀	64	20	6	3	1			1		95	.55

Although marked fluctuations occur, they are inconsistent and the differences are not certainly significant. Males born in the three autumn months, for example, show the highest average grade, while females born during the same three months display the lowest. Hence, it is probable that season of birth has little if any effect on grades of spotting.

SUMMARY

In an inbred strain of mice a white ventral patch ranging in size up to 10 percent of the ventral surface is present in a majority of the animals. These patches are not only more frequent in males than in females but average larger as well.

The presence of white ventral spotting seems to be due neither to a single dominant nor recessive gene. The occurrence and extent of these markings, however, do appear to have an hereditary basis. In this strain of mice, the three chief lines show a tendency towards differentiation in the amount of spotting present. The coefficient of correlation between mid-parental grade and grade of offspring is +0.258. When the method of partial correlation is employed, the value of r for grade of sire and grade of offspring (with dam held constant) is +0.243; that for grade of dam and grade of offspring (with sire held constant), +0.086. From these figures, it ap-

pears that the sire has more influence on the grade of offspring than does the dam. The total effect of heredity is relatively slight, most of the variation being non-genetic in origin.

The age of dam or sire apparently has a very slight influence in determining spotting since the coefficient of correlation is but +0.105. Birth rank and season of year likewise have little influence; so most of the non-genetic variation, whether it be due to accidents of development or to other causes, is still unaccounted for.

LITERATURE CITED

ALLEN, G. M., 1914 Pattern development in mammals and birds. Amer. Nat. **48**: 385–412.

DUNN, L. C., 1920 Types of white spotting in mice. Amer. Nat. **54**: 465–495.

1925 The isolation of a pattern variety in piebald house mice. Amer. Nat. **59**: 36–49.

KEELER, C. E., 1931 A new mutation to "dominant spotting" (W) in the house mouse. J. Hered. **22**: 273–276.

LITTLE, C. C., 1917 Evidence of multiple factors in mice and rats. Amer. Nat. **51**: 457–480.

1924 The genetics of spotting. Carnegie Instn. Washington Year Book **23**: 42.

1926 The inheritance of blaze spotting in mice. Anat. Rec. **34**: 171.

WRIGHT, S., 1926 Effects of age of parents on characteristics of the guinea pig. Amer. Nat. **60**: 552–559

INHERITANCE IN NICOTIANA TABACUM. XIII. THE CYTOGENETICS OF "DEFORMED," AN X-RAY DERIVATIVE[1]

T. H. GOODSPEED AND P. AVERY
University of California, Berkeley, California

Received February 6, 1933

At the present time attention is being directed to the genetics and cytology of conditions arising from spontaneously occurring and induced mosaicism. Where the situation is relatively simple, correspondence of cytological and genetic evidence is obtainable (MOHR 1932). In other cases greater or lesser complexity is involved and the correspondence may not be clearly shown (STEIN 1930, 1932). The complexity in the case under discussion here arises primarily from the fact that we are dealing with an X-ray induced physical attachment of chromosomes. This results in somatic elimination of the products of attachment and also in a series of breakages of the physical union which are reflected in the production of numerous by-products. A consistent cytogenetic interpretation of the situation involved is possible primarily because the "fluted" chromosome, which has already been studied genetically, is involved.

An earlier paper in this series (GOODSPEED 1930) dealt with some products of X-ray and radium treatment of sex cells of *Nicotiana tabacum* var. *purpurea*, which behaved genetically like transgenations. In recent reports (GOODSPEED 1931), the occurrence of at least one of these induced character changes is reinterpreted in the light of additional cytological evidence, as the result of quantitative rather than qualitative alteration. A résumé has recently been given (GOODSPEED 1932) of the results of the various types of cytogenetic investigations under way in the UNIVERSITY OF CALIFORNIA BOTANICAL GARDEN on the categories of alteration in the germ-plasm of *N. tabacum* induced by high frequency radiation. Reference is there made to a morphological type called "deformed," which appeared in X_1 and has been the subject of extended genetic and cytological studies, the results of which are herein reported. The designations X_1, X_2, X_3, etc., and R_1 (STEIN 1932), R_2, R_3 et cetera, have been used throughout these investigations to signify the successive generations derived from X-ray or radium treatment of reproductive or meristematic tissues. Thus, for example, an X_1 plant is one produced from an X-rayed seed, or from an X-rayed growing point of a seedling, or from a zygote produced by the

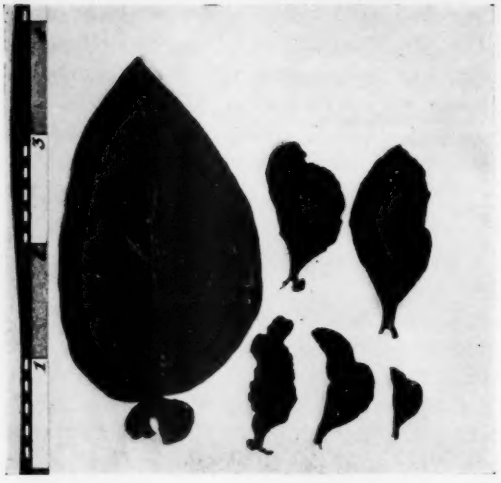
[1] The investigations reported on here have been aided by grants from the Board of Research of the UNIVERSITY OF CALIFORNIA, and from the Committee on the Effects of Radiation of the NATIONAL RESEARCH COUNCIL.

union of sex cells one or both of which have been subjected to X-radiation.

Appropriate X-ray treatment of EMC and PMC (PMC = pollen mother cells; EMC = embryo-sac mother cells) of *tabacum* in active meiotic stages is always productive of a high degree of variation in expression of all vegetative and floral characters in X_1 (GOODSPEED 1929). This extended range of alteration in expression is to be seen not only in the immediate progenies but also in subsequent generations following treatment. Variation in form, size, and color of flowers and leaves occurs along with variation in height, habit, and time of flowering, et cetera, but only rarely are deformities due

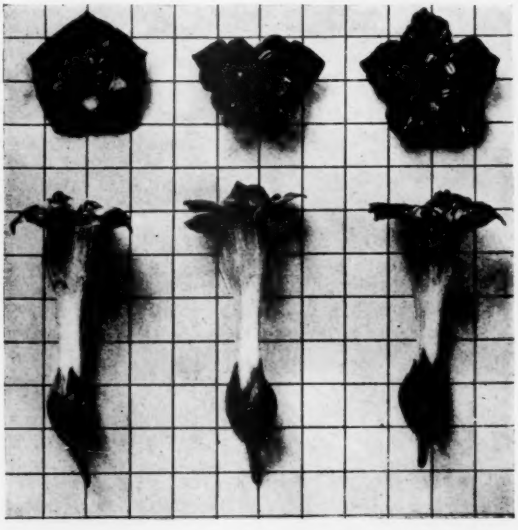
FIGURE 1.—Young *deformed* plant, X_4 (31.212P55), showing abnormalities in tissue development, similar to those seen in the original *deformed* (28.303P51).

to tissue degeneration to be observed. In the case under discussion such a condition is involved. The original X_1 plant (28.303P51) was the result of using normal pollen on a flower which had been subjected to X-radiation (GOODSPEED 1929, for treatment) when it was almost at anthesis. At this stage the EMC are in the meiotic condition. None of the other plants resulting from this cross were as abnormal in appearance as was 28.303P51. From the seedling stage (figures 1 and 6) it was peculiar structurally, with imperfect, asymmetric venation, which made the leaves curl or sometimes allowed them to develop on one side only of the midrib (figure 2). The flowers were flecked with white and much dissected, making the limb very irregular in size and shape (figures 3 and 7, e, f). The anthers



a b

FIGURE 2—a, Leaf from *control*, *N. tabacum* var. *purpurea;* b, Five leaf types from an X_3 *deformed* (29.137P51).



a b c

FIGURE 3—a, Flower from *control*, *N. tabacum* var. *purpurea;* b and c, Flowers from X_1 *deformed*, showing the white-flecked and dissected limb characteristic of this type.

were of very different types and sizes but contained some viable pollen and dehisced normally. Some seed was set but often buds were abscissed at a very young stage of development. The flower was approximately equal to control in length (55 mm) when best developed but was only about 25 mm in width of limb. The following terminology is here employed in referring to major distinctions in phenotypic expression:

control (*cn*) refers to *Nicotiana tabacum* var. *purpurea*, grown in the pure line (figures 2, a and 7, a);

normal (*n*) refers to plants in lines derived from *deformed* phenotypically equal to *control;*

deformed (*d*), a type characterized by abnormalities(X-ray induced) in tissue production and alignment, the cytogenetics of which is here described (figures 1; 2, b; 3; 5; 6; 7, e, f);

fluted (*f*), a distinct character complex elsewhere described (CLAUSEN and GOODSPEED 1926) and involving reduction in flower size, fluting of corolla limb, and other morphological distinctions from *control* (figure 7, b);

mammoth (*m*), a type characterized by increase in height accompanied by shortening of internodes and delayed time of blooming (figure 5);

long-flowers (*l*) refers to a series of types possessing distinctly increased length of flower. This designation is used to distinguish these derivatives from types already described (CLAUSEN 1931, GOODSPEED 1932) which possess increased flower-size (figure 7, c, d).

The character of the tissue abnormalities peculiar to *deformed* suggests, at first glance, the effects of some such disease as "mosaic" (figures 1 and 2, b). On the other hand, the cytogenetic evidence shows that a diseased condition is not involved and that *deformed* is transmitted through a chromosomal mechanism in which elimination, in mitosis, of chromosomes or parts of chromosomes is concerned. In addition, inoculation tests using unfiltered crude extract from *deformed* plants as the inoculum and vigor-

TABLE 1

Derivation of progenies from deformed.

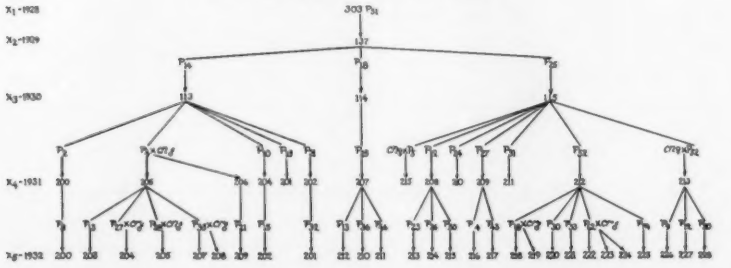


TABLE 2*

Classification of progenies from deformed.

POPULATION	PARENTAL PHENOTYPE	*n*	*d*	*f*	*l*	*nl*	*m*	*df*	*dl*	*dm*	*fm*	*lm*	*crf*	*ccv*		NEW TYPES	*ntc*	TOTAL	TOTAL			
														n	*l*				*d*	*f*	*l*	*m*
29.137	*d*	6	30	5	7	..	2	..	..	..	..	..	..	..	..	..	..	50	30	5	7	2
30.113	*d*	6	16	4	2	..	..	1	2	..	..	..	..	..	..	..	..	31	19	5	4	..
30.114	*f*	11	..	37	..	..	..	..	..	..	..	..	..	..	..	..	1	49	..	37	..	..
30.115	*d*	4	17	15	9	..	..	..	4	1	..	1	..	..	..	..	..	51	22	15	14	2
31.200	*f*	26	..	22	..	..	..	..	..	..	..	..	..	..	..	..	2	50	..	22	..	..
31.201	*df*	..	..	..	..	..	16[a]	..	..	19[b]	11	4	..	..	..	..	..	50	19	..	..	50
31.202	*l*	3	2	11	14	7	2	..	3	..	..	3	..	..	..	..	..	45	5	11	27	5
31.204	*n*	46	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	48	..	..	..	..
31.205	*d*	5	13	9	3	..	1	..	..	2	1	1	..	..	..	..	..	35	15	10	4	5
31.206	*d* ♀ × *cn* ♂	11	..	8	3	..	..	..	..	..	..	..	..	..	..	..	2	24	..	8	3	..
31.207	*f*	30	..	18	..	..	..	..	..	..	..	..	..	..	..	..	..	48	..	18	..	..
31.208	*l*	16	..	2	26	4	1	..	1	..	..	..	..	..	..	..	..	50	1	2	31	1
31.209	*dl*	17	..	12	20	..	..	..	..	..	..	..	..	..	..	..	..	49	..	12	20	..
31.210	*f*	10	..	43	..	..	..	..	..	..	..	..	..	..	..	..	2	55	..	43	..	..
31.211	*dl*	46	..	..	..	..	..	..	..	..	..	..	..	..	..	..	4	50	..	..	..	..
31.212	*d*	6	25	14	11	..	4	..	3	..	2	4	..	..	..	..	..	69	28	16	18	10
31.213	*cn* ♀ × *d* ♂	2	..	1	19	2	..	..	..	..	..	..	..	..	..	..	1	25	..	1	21	..
31.215	*cn* ♀ × *d* ♂	12	..	3	8	..	..	..	..	..	..	..	..	..	..	..	3	26	..	3	8	..
32.200	*ccv*	12	..	3	1	..	..	1	..	..	12[c]	..	..	..	..	15[d]	6	50	..	..	..	..
32.201	*dl*	18	..	..	..	..	..	..	..	..	..	..	..	..	..	10[e]	3	31	..	..	..	..
32.202	*n*	47	..	..	..	..	..	..	..	..	..	..	..	..	..	..	3	50	..	..	..	..
32.203	*l*	23	..	..	16	10	..	..	..	..	..	..	..	..	..	..	..	49	..	..	26	..
32.204	*d* ♀ × *cr* ♂	30	..	..	1	..	..	..	..	..	..	..	3	14	1	..	..	49	..	3	2	..
32.205	*d* ♀ × *cn* ♂	13	..	13	2	..	..	..	..	..	..	..	..	..	..	..	6	34	..	13	2	..
32.207	*d*	1	14	3	5	..	..	10	6	..	..	..	..	..	..	..	..	39	30	13	11	..
32.208	*d* ♀ × *cr* ♂	2	..	..	2	..	..	..	..	..	..	..	25[f]	9	5	..	..	43	..	24	7	..
32.209	*l* in F_1 *d* ♀ × *cn* ♂	18	..	..	27	5	..	..	..	..	..	..	..	..	..	..	..	50	..	..	32	..
32.210	*crf*	22[g]	..	27[g]	..	..	..	..	..	..	..	..	..	..	..	..	..	49	..	27	..	..

TABLE 2 (Continued)

POPULATION	PARENTAL PHENOTYPE	*n*	*d*	*f*		*sl*	*m*	*df*	*dl*	*dm*	*fm*	*lm*	*crf*	*ccv*		NEW TYPES	*vts*	TOTAL	TOTAL			
														n	*l*				*d*	*f*	*l*	*m*
32.211	*f*	24	..	23	..	..	..	..	..	..	..	..	..	..	..	..	3	50	..	23	..	..
32.212	*n* in *f* line	47	..	2	..	..	..	..	..	..	..	..	..	..	..	..	1	50	..	2	..	..
32.213	*l*	21	..	2	11	4	..	..	..	..	..	..	..	..	..	..	6	44	..	2	15	..
32.214	*l*	15	..	10	13	1	1	..	..	..	..	..	..	..	..	..	3	43	..	10	14	1
32.215	*sl*	33	..	2	12	3	..	..	..	..	..	..	..	..	..	..	..	50	..	2	15	..
32.216	*n* in *l* line	50	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	50	..	..	..	..
32.217	*l*	17	..	6	24	..	..	..	..	..	..	..	..	..	..	..	2	49	..	6	24	..
32.219	*d* ♀ × *cr* ♂	6	..	..	1	..	..	..	..	..	..	..	5	2	1	..	1	16	..	5	2	..
32.220	*n*	34	..	..	..	..	15	..	..	1	..	..	..	..	..	..	..	50	..	..	..	16
32.221	*ml*	1	..	1	5	..	..	..	..	..	..	1	..	..	..	..	..	8	..	1	6	1
32.222	*ccv*	9	2	1	2	..	..	..	..	..	..	..	..	6	..	..	..	20	2	1	2	..
32.223	*ccv* ♀ × *cr* ♂	3	..	..	7	..	..	..	..	..	..	..	34	2	3	..	..	49	..	34	10	..
32.224	*ccv* ♀ × *cr* ♂	10	..	..	6	..	..	..	..	..	..	..	23	1	2	..	1	43	..	23	8	..
32.225	*n*	50	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	50	..	..	..	..
32.226	*n* in F_1 *cn* ♀ × *d* ♂	44	..	..	..	..	6	..	..	..	..	..	..	..	..	..	..	50	..	..	..	6
32.227	*l* in F_1 *cn* ♀ × *d* ♂	26	..	10	11	..	..	..	..	..	..	..	..	..	..	..	2	49	..	10	11	..
32.228	*sl* in F_1 *cn* ♀ × *d* ♂	14	..	8	10	3	..	..	..	..	..	..	..	..	..	10[e]	4	49	..	8	13	..
Total		847	119	315	278	39	48	12	18	24	26	14	90	34	12	35	58	1969	171	415	357	99

[a] Classification into *fluted* and *long-flowers-mammoth* not completed because of delayed blossoming.
[b] Some of these were *deformed-fluted* and *deformed-long-flowers-mammoth*.
[c] These mammoth have been classed as *fluted*, but their delayed blossoming has prevented verification.
[d] See description.
[e] See description.
[f] One plant with coral flowers was not *fluted*.
[g] All with coral flower color.
[h] *Abbreviations used*: *ccv* = carmine-coral variegation; *cn* = control; *cr* = coral; *d* = deformed; *f* = fluted; *l* = long-flowers; *m* = mammoth; *n* = normal; *sl* = super-long-flowers; *vts* = variants.

ously growing young plants of *N. tabacum* (Commercial Havana No. 38) as test plants gave negative results after holding for 30 days. We are indebted to Dr. W. TAKAHASHI of the Division of Plant Pathology, UNIVERSITY OF CALIFORNIA, for carrying out these tests. In view of the negative evidence which they gave and the character of the genetic evidence, it did not appear necessary to test the possibility of transmission following grafting. Apparently *deformed* is a condition comparable in its expression to STEIN's "farb- und formdefekt" types (STEIN 1926) and her "Phytocarcinome" (STEIN 1932) for it involves a mosaicism of light and dark areas in the leaves as well as other evidence of irregular tissue development (figure 1).

MATERIALS AND METHODS

The preceding chart (table 1) traces the ancestry of our present *deformed* plants to the original X_1 plant (28.303P51) already described. Approximately 2000 plants (table 2) have been grown under the designations given in this chart, and more than 100 have been studied cytologically.

In addition to 28.303P51, a number of other plants exhibiting deformities of various types have appeared in X_1 or in later generations. While we do not feel justified in considering each of these reappearances as equivalent to the original *deformed*, nevertheless all our types are undoubtedly reflections of somatic chromosome elimination.

Most of the cytological study of plants in the *deformed* group was made on meiotic divisions in PMC. EMC were also used, and root tips were examined to obtain evidence as to the chromosome behavior in somatic mitoses. For PMC, iron-aceto-carmine smears were found most satisfactory. Permanent brazilin and aceto-carmine slides were made, following WEBBER'S (1929) method, and STEERE'S (1931) modification of MCCLINTOCK'S method. Anthers and ovaries showing meiotic stages were also fixed in Navashin's fluid, the anthers being first dipped in Carnoy's solution. Root tips were fixed in Navashin. Paraffin sections were stained with iron-alum haematoxylin or crystal violet. The drawings (figures 10–23) were, as indicated in the legends, made from slides prepared according to the various techniques just mentioned. The differences in magnification noted are referable to the effects of the various techniques on chromosome size.

GENETICS OF DEFORMED

A striking genetic characteristic of all *deformed* plants so far tested has been their consistent production of progenies containing plants belonging to five or more distinct morphological types (table 3 and figure 4). Plants of each of these types derived from *deformed* have become the sources of

races which differ significantly in their genetic behavior. All of these races have been characterized by continued segregation of types referable to quantitative variation in the *fluted*, or F chromosome. The X_3 progeny from *deformed* shown in figure 5 contains such a series of types.

Deformed plants transmit the *deformed* condition to approximately half their progeny upon selfing, as shown in table 3.

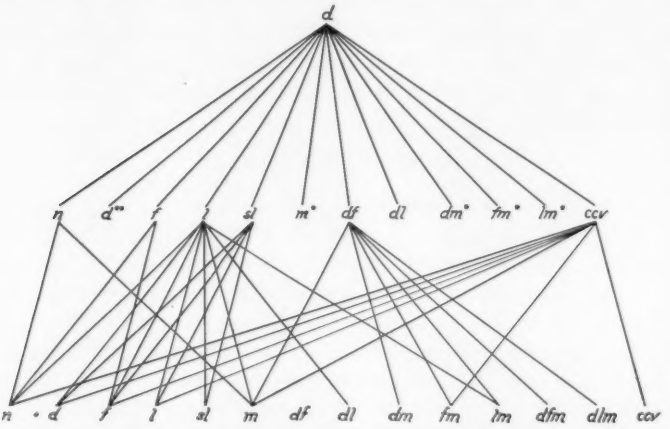


FIGURE 4.—Transmission of types derived from *deformed*. (* Segregation of types in progenies of *mammoth* has not been studied. ** *Deformed* segregates as before.)

TABLE 3

Transmission of deformed.

POPULATION	n	d	f	l	m	df	dl	dm	fm	lm	TOTAL	TOTAL			
												d	f	l	m
29.137	6	30	5	7	2	..	..	..	..	..	50	30	5	7	2
30.113	6	16	4	2	..	1	2	..	..	..	31	19	5	4	..
30.115	4	17	15	9	..	..	4	1	..	1	51	22	15	14	2
31.205	5	13	9	3	1	..	..	2	1	1	35	15	10	4	5
31.212	6	25	14	11	4	..	3	..	2	4	69	28	16	18	10
32.207	1	14	3	5	..	10	6	..	..	..	39	30	13	11	..
Total	28	115	50	37	7	11	15	3	3	6	275	144	64	58	19

Thus in the X_2 progeny of 50 plants grown from the original *deformed* plant there were 30 more or less *deformed* plants. A *deformed* plant of this X_2 population gave 31 plants in X_3, 19 of which were *deformed*. One of these X_3 *deformed* plants gave an X_4 of 35 plants, 15 of which were *deformed*, and one of these in turn gave an X_5 of 39 plants, 30 of which showed deformity. When we combine with these four populations the two others from *de-*

formed plants, the total number of *deformed* is approximately one-half of all the plants grown (144 in 275).

This transmission of *deformed* suggests that of a quantitative chromosome variation in that no pure-breeding *deformed* occur; and since *deformed* does not segregate in Mendelian ratios in F_2 of *deformed*×*control*, it is clear that

FIGURE 5.—Progeny of a *deformed* (30.115), showing two *deformed* ("x") in the foreground, and a *long-flowers-mammoth* ("y") in the background.

a monogenic mutation is not involved. Further, reference to tables 3 and 5 shows that *deformed* is not transmitted as a simple monosomic or trisomic condition, since *deformed*×*control* gives no *deformed*. It is to be noted, however, that like these unbalanced types, *deformed* is transmitted to approximately 50 percent of its progeny. As will be shown in the discussion of the cytological evidence, this situation is produced by the operation of a mechanism involving chromosome attachment.

It is to be noted that the expression of the *deformed* condition does not become less pronounced in succeeding generations. It varies greatly in its expression in different individuals in the same population and in different parts of the same individual, but some of the plants in X_5 were even more deformed than the original X_1 plant (figure 6).

Deformed plants give *long-flowers*, *fluted*, and *mammoth* in their progenies and these may appear in combination with the *deformed* condition or with each other, to give the classes *deformed-fluted*, *deformed-long-flowers*, *deformed-mammoth*, *fluted-mammoth*, *long-flowers-mammoth*, *deformed-fluted-mammoth*, and *deformed-long-flowers-mammoth* (figures 4 and 7). The classification of the progenies (table 3) of these *deformed* plants shows, in the first place, the great variety of forms obtained and, in the second place, the comparative similarity in the ratios of the types to one another in each population. In these populations the number of *long-flowers* is approximately equal to the number of *fluted*, and taking all the populations together, there are 58 *long-flowers* to 64 *fluted*. Of the total 275 plants, 144 or 52 percent are *deformed;* 58 or 21 percent are *long-flowers;* 64 or 23 percent are *fluted;* 19 or 7 percent are *mammoth;* and 28 or 10 percent are *normal*.

FIGURE 6.—Young seedlings in lines derived from *deformed:* 32.200, a population showing no *deformed*, and 32.207, a population in which a large number of the seedlings are *deformed*.

The classes of plants obtained in these progenies show clearly that the *fluted* (F) chromosome is present in different doses, for most of these types have been obtained under natural conditions when the F chromosome was

TABLE 4

Table of flower lengths in deformed populations (in mm).

POPULATION	FLUTED							NORMAL											LONG-FLOWERS					SUPER-LONG FLOWERS			TOTAL
	40	41	42	43	44	45	46	47	48	49	50	51	52	53	54	55	56	57	58	59	60	61	62	63	64	65	
29.137	..	..	1	1	..	1	..	..	1	..	2	3	4	..	7	8	4	..	..	..	..	2	1	..	1	1	37
30.113	..	..	2	1	2	..	..	..	..	1	3	1	..	..	1	1	..	..	..	..	..	2	1	..	..	..	15
30.115	..	4	4	3	1	4	..	1	..	1	2	3	2	..	1	1	1	1	1	1	2	6	..	..	..	3	42
31.205	1	1	1	4	1	..	1	..	1	1	2	1	3	1	..	1	..	..	..	..	1	..	1	..	..	..	21
31.212	3	2	5	5	1	1	1	..	..	1	2	2	3	3	2	..	3	3	..	1	3	1	2	..	..	2	46
32.207	..	3	3	3	2	3	..	..	1	2	..	1	1	1	1	2	2	2	1	..	..	..	1	..	..	1	30
Total	4	10	16	17	7	9	2	1	3	6	11	11	13	5	12	13	10	6	2	2	6	11	6	..	1	7	191

present in abnormal number or constitution (CLAUSEN 1931). However, the ratios (table 3) in which these types occur here cannot be explained according to the genetic interpretations previously postulated in the case of un-X-rayed plants of the "*fluted* assemblage" (see CLAUSEN, l. c.). The unusual ratios obtained here point to the operation of a mechanism not previously encountered. Thus, the occurrence of both *fluted* and *long-flowers* plants in the progeny of *deformed*, a member of the *fluted* assemblage, is unexpected. *Fluted* and *long-flowers* appear in approximately equal numbers in such progenies, whereas, in un-X-rayed lines, *fluted* (monosomic F) gives *fluted* and *normal* (see CLAUSEN, l. c.) and *large-lax* (trisomic F) gives large flowered plants and *normal* in selfed progenies. Flower lengths in populations from *deformed* are given in table 4. The situation detailed there illustrates the F chromosome classes which occur.

Evidence that *deformed* produces gametes which vary quantitatively as regards the F chromosome from the nullo- to the triplo-F condition is furnished by the crosses of *deformed* × *control*. It is significant that in these crosses no *deformed* have occurred. In comparison to the progenies obtained by selfing the same *deformed* plants, these populations show only a few classes of individuals: chiefly *normal*, *long-flowers*, and *fluted* (table 5). Two populations from *deformed* ♀ × *control* ♂ (31.206 and 32.205) and two populations of the reciprocal cross, but involving other *deformed* plants (31.213 and 31.215) have been grown. In each case more *fluted* were obtained when *deformed* was used as the female parent and more *long-flowers* when used as the male parent. This indicates that nullo-F female gametes are not as effectively eliminated by competition as are nullo-F male gametes. It is to be noted that where *deformed* was pollen parent, the progeny contained *fluted* and a large number of *long-flowers*, whereas these monosomic and trisomic conditions in un-X-rayed lines are transmitted only to a very small percentage through pollen (see CLAUSEN, l. c.). The population 31.213 is especially significant in this connection in that 21 of the total 25 plants were *long-flowers*. Here, *long-flowers* must be the result of transmission of excess chromosome material by the pollen of *deformed*.

TABLE 5

Progenies of deformed crossed with control.

POPULATION	CROSS	*n*	*d*	*f*	*l*	*tls*	TOTAL
31.206	*d* ♀ × *cn* ♂	11	..	8	3	2	24
31.213	*cn* ♀ × *d* ♂	2	..	1	21	1	25
31.215	*cn* ♀ × *d* ♂	12	..	3	8	3	26
32.205	*d* ♀ × *cn* ♂	13	..	13	2	6	34
Total		38	0	25	34	12	109

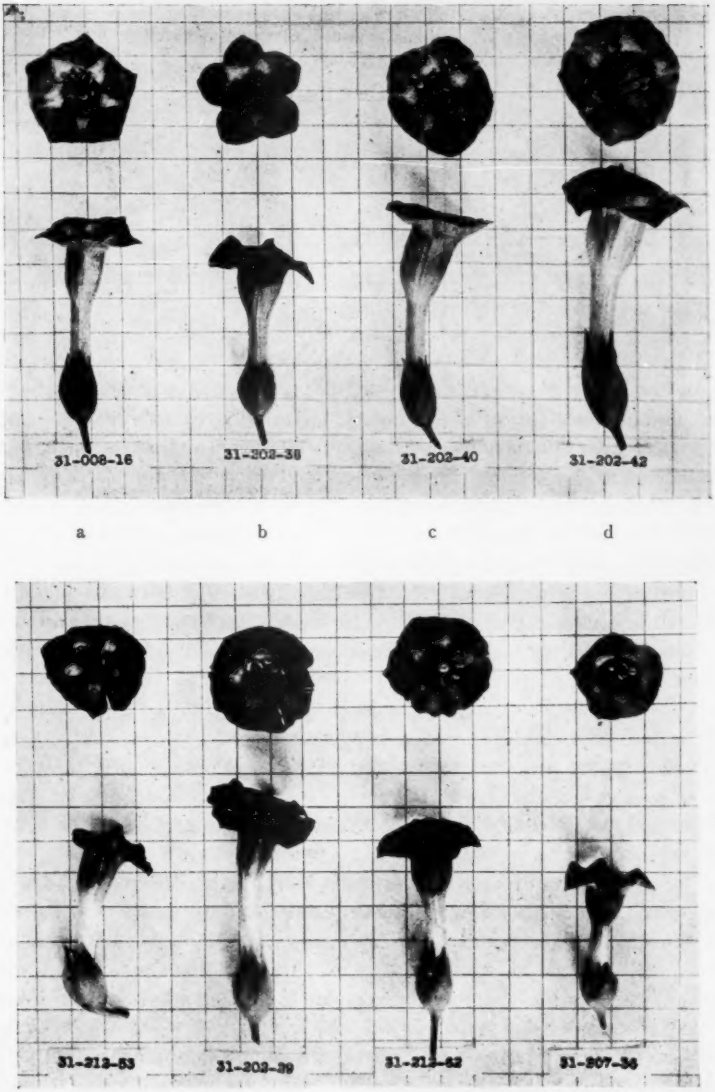


FIGURE 7.—Flowers of *control* and of lines derived from *deformed:* a, *control;* b, *fluted;* c, *long-flowers;* d, *super-long-flowers;* e, *deformed;* f, *deformed-long-flowers;* g, *carmine-coral* variegation; h, *coral-fluted.*

TABLE 6

Progenies of deformed and carmine-coral crossed with coral.

POPULATION	CROSS	n	d	l	crf	ccv n	ccv l	ccv ns	TOTAL
32.204	d♀ × cr♂	30	..	1	3	14	1	..	49
32.208	d♀ × cr♂	2	..	2	25*	9	5	..	43
32.219	d♀ × cr♂	6	..	1	5	2	1	1	16
32.223	ccv♀ × cr♂	3	..	7	34	2	3	..	49
32.224	ccv♀ × cr♂	10	..	6	23	1	2	1	43
Total		51	0	17	90	28	12	2	200

* One plant with coral flowers was not *fluted*.

The fact that *coral* (CLAUSEN, l. c.) behaves genetically as a Mendelian recessive borne by the F chromosome has made it possible to analyse further the constitution of the functional gametes of *deformed* by means of crosses with homozygous *coral* plants. The results of three such crosses are shown in table 6. Where the F chromosomes of *deformed* have been eliminated from the gametes or zygotes, F_1 is *coral-fluted*, that is, it exhibits the morphological characters of *fluted* together with coral flower color. The number of *coral-fluted* plants thus becomes an index to the amount of F chromosome elimination. In the plants which are not *coral-fluted*, the occurrence of *carmine-coral* variegation indicates that somatic elimination of the F chromosome derived from the *deformed* parent is going on (figure 7, g). The type of variegation here is similar to that reported by CLAUSEN (1930) as due to elimination in mitosis of a fragment of the F chromosome. The degree of expression of *carmine-coral* variegation in different plants is highly variable, some showing only small coral flecks while others have whole branches bearing only coral flowers. As to flower size, the plants which show this variegation may be either *normal* or *long-flowers*.

The *coral* originally used in these crosses was not a *deformed* derivative, but more recently a *coral* race has been established in the *deformed* lineage. As shown in tables 1 and 2, 31.207 is a *fluted* line derived from an X_2 plant, and P36 of this culture was a small, *fluted* plant which bore only coral flowers (figure 7, h). From it a culture (32.210) has been obtained consisting of *normal-coral* and *coral-fluted* plants. In many other instances it has been clear that the alteration of the F chromosome productive of coral flower color has occurred, for plants in populations derived from *deformed* and into whose ancestry no *coral* has been introduced, have shown *carmine-coral* variegation. Thus 31.212P62 was a non-*deformed* plant of normal flower length (listed as *normal* in table 2), whose flowers showed considerable *carmine-coral* variegation (figure 7, g). It was selfed and crossed

twice with *coral*, different branches being used in each case. As is evident from table 6, the progenies resulting from these crosses with *coral* (32.223 and 32.224) resemble closely those resulting from crosses of *deformed* with *coral*. The selfed progeny also suggests that of *deformed*, for two typical *deformed* plants appeared in the progeny of this non-*deformed*. There were also several plants in this population which again showed *carmine-coral* variegation but no deformity, so that a somatic loss leading to a condition other than that responsible for *deformed* must have occurred.

From these observations concerning the genetic behavior of *deformed* when selfed and crossed with *control* and with *coral*, it is evident that:

(1) The *deformed* condition is transmitted from one generation to the next through selfing but not through crosses with the control.

(2) The mosaic and variable expression of tissue abnormality characteristic of *deformed* resembles the color variegation produced on crossing *deformed* with *coral* and indicates that diversity in the chromosome constitution of somatic cell lines is responsible for both of these expressions.

(3) Progenies of *deformed* selfed and crossed with *coral* consist of many distinct types, while in progenies from crosses with *control* there are relatively few distinct types.

There are apparently some significant differences in the genetic behavior of *fluted* and *long-flowers* derived from *deformed*. Thus *fluted* selfed gives the two classes expected, *normal* and *fluted*, while the progenies of *long-flowers* usually consist of three to seven classes and contain *fluted* as well as *long-flowers*. *Fluted* has never given *deformed*, while *long-flowers* has given *deformed*. These facts indicate that the segregation of *fluted* is accompanied by the loss of ability to produce the *deformed* condition and to produce progenies consisting of many distinct types, while *long-flowers* may retain both of these capacities.

There is a considerable difference in the number of *fluted* from *fluted* selfed (table 7), but the average of all the populations grown is so close to that found in untreated lines that this difference probably has no sig-

TABLE 7

Transmission of fluted from deformed.

POPULATION	*n*	*f*	*n/2*	TOTAL	PERCENT *f*
30.114	11	37	1	49	76
31.200	26	22	2	50	44
31.207	30	18	..	48	38
31.210	10	43	2	55	78
32.210	22	27	..	49	55
32.211	24	23	3	50	46
Total	123	170	8	301	56 (average)

nificance, especially since the percentage of *fluted* often varies widely in different untreated cultures. (The percentages here varied from 38 to 78, but the average of 6 populations was 56 percent, which is close to the percentage reported by CLAUSEN and GOODSPEED [1926] for the transmission of the *fluted* character in untreated cultures.) One exception to the usual mode of transmission of *fluted* occurred in the progeny (31.201) obtained from *deformed-fluted* (table 2). Here, instead of approximately one-half *fluted* and one-half *normal*, the whole progeny consisted of *mammoth*. Due to failure of these plants to blossom during the normal flowering period, their classification as regards flower-size into *long-flowers*, *fluted*, and *normal* was not completed. It was evident, however, that these three types did occur. Of 50 plants, 19, or a little less than half, were deformed, and some of these were also *long-flowers* or *fluted*. Here, the types appearing in connection with the *mammoth* character are the same as those in non-*mammoth* populations from *deformed*. Apparently the modification of the F chromosome necessary to produce the *mammoth* condition must have occurred in the *deformed-fluted* parent at some time before sex-cell formation, but after the character of the plant itself had already been determined by the non-*mammoth* producing F chromosome.

The same change in the F chromosome which led to the production of all *mammoth* in 31.201 must also occur in *normal* (not *fluted*) *deformed* plants since *mammoth* occurs in many progenies from *deformed*. Undoubtedly plants heterozygous for *mammoth* occur in these progenies but due to the complexity of types dealt with, they have not been classified with certainty, so that *mammoth* has only been dealt with when it has segregated in a homozygous state. Two exceptional *mammoth* types were included among the ten *mammoth* in 31.212. These plants had the characteristic growth habit of *mammoth* with the leaf type of *long-flowers-mammoth*, and were only distinct from other *mammoth* when they bloomed late in the season but several months before *normal-mammoth*. Both of these plants were long-flowered and their abnormal behavior in respect to the flowering season is possibly related thereto. If *long-flowers* is due to the presence of supernumerary F chromosomes and *mammoth* to alteration of this chromosome, these plants may have possessed two F's carrying the *mammoth* modification and one which did not, the result being a condition approaching *mammoth* but altered in the direction of *normal* as regards time of blooming. The progeny of one of these plants (32.221) bears on this assumption, for of 8 plants grown, 1 was *mammoth* and 5 were *long-flowers*.

Segregation for *mammoth* in two X_3 cultures in three to one ratios, indicates that the change in the F chromosome responsible for the *mammoth* condition had been produced in X_3 and transmitted to the parent plants which were undoubtedly heterozygous for *mammoth*. Thus 32.220 came

from a *normal* in a population from *deformed*, and consisted of 50 plants, 16 of which were *mammoth*. In the case of 32.200, which will be discussed later, 12 out of 50 plants were *mammoth*. The X_3 parent of this X_5 population was *fluted*, so that the change in the F chromosome responsible for the production of *mammoth* did not occur in the X_2 *deformed* plant, but probably must have occurred following its presence in the grandparental zygote, since the parent plant was of *normal* habit and flower size. Six *mammoth* occurred among the 50 of 32.226, which was the F_2 of a *normal* F_1 from *control*×*deformed*. Thus *mammoth* is constantly being produced and constantly segregating from *deformed* and in genetic behavior resembles a recessive gene mutation.

As already mentioned, the genetics of the long-flowered types is not as simple as that of *fluted* derived from *deformed*. The mode of origin of *long-flowers* as well as its subsequent behavior indicates that it is a reflection of the presence of at least a part of the F chromosome in excess of its normal diplo-condition. CLAUSEN (1931) has shown that triplo-F is a large-flowered type, morphologically close to the trisomic "enlarged," and he calls this form "*large-lax*". Our *long-flowers* is close to *large-lax* in its morphological features but genetic tests have not been made which identify the former type with the F chromosome. It is possible that some other chromosome, such as the *enlarged* chromosome, may be concerned in the production of at least some of the long-flowered types derived from *de-*

TABLE 8

Transmission of long-flowers types.

POPULATION	n	d	f	l	al	m	dl	ml	tls	TOTAL	TOTAL d	TOTAL f	TOTAL l	PERCENT l
31.202	3	2	11	14	7	2	3	3	..	45	5	11	27	60
31.208	16	..	2	26	4	..	1	1	..	50	..	2	32	64
31.209	17	..	12	20	..	..	..	..	..	49	..	12	20	41
32.203	23	..	..	16	10	..	..	..	..	49	..	..	26	53
32.213	21	..	2	11	4	..	..	..	6	44	..	2	15	34
32.214	15	..	10	13	1	1	..	..	3	43	..	10	14	33
32.215	33	..	2	12	3	..	..	..	..	50	..	2	15	30
32.217	17	..	6	24	..	..	..	..	2	49	..	6	24	49
32.209*	18	..	..	27	5	..	..	..	..	50	..	..	32	64
32.227*	26	..	10	11	..	..	..	..	2	49	..	10	11	22
32.228*	24**	..	8	10	3	..	..	..	4	49	..	8	13	27
Total	213	2	63	184	37	3	4	4	17	527	5	63	229	43 (average)

* Progeny of F_1 *d* ♀ × *cn* ♂.

** 10 were somewhat longer than normal, but did not belong to the *long-flowers* class.

formed. We believe, however, that the genetic analysis is adequate and simpler, if these types are assumed to be produced by the presence of excess F chromosome material.

As is shown in table 8, 9 of the 11 progenies of *long-flowers* so far grown have contained *fluted* as well as *long-flowers*, which is unexpected since monosomics are rare in progenies of trisomics. The progenies obtained from *long-flowers* suggest those obtained from *deformed*, for occasionally *mammoth* and *deformed* as well as *long-flowers* and *fluted* occur. The percentage of *long-flowers* from *long-flowers* selfed varies from 22 to 64, the average for all populations being 43 percent (table 8). The plants in this *long-flowers* class fall into at least two distinct flower-length groups: the first 58-62 mm long; the second, *super-long-flowers*, 63-70 mm long. Three populations contained no *super-long-flowers* but did contain a considerable number of *fluted*. On the other hand, two populations contained no *fluted* but higher proportions of *super-long-flowers* than did the other populations. (31.202 contained a higher proportion of *super-long-flowers* together with a considerable number of *fluted*. The genetic and cytological evidence indicates that while the parent of this population was phenotypically *long-flowers*, the genotype transmitted through the gametes was that of a *deformed*.)

Super-long-flowers resembles *super-enlarged* in flower length but differs from it in other morphological characters. *Super-enlarged* is known to be a tetrasomic form and has been reported by CLAUSEN and GOODSPEED (1924) to give 20 percent of *super-enlarged* and 77 percent of *enlarged* in its progeny. It is therefore significant that *super-long-flowers*, 31.208P50, gave no more *super-long-flowers* in its progeny (31.215) than did other *long-flowers* not of the super-long class. It also gave two *fluted*. A *super-long-flowers* in F_1 of *deformed*×*control* also showed this same type of behavior, giving 3 *super-long-flowers*, 10 *long-flowers*, and 8 *fluted* in a population of 49 plants. These facts indicate that while *super-long-flowers* may in some cases represent a tetrasomic type, in other cases it is not due to the presence of four identical and independent chromosomes.

In certain populations, *long-flowers* shows considerable variation as to habit and flower-width, but this difference is not associated with differences in genetic behavior. Thus three categories of *long-flowers* in 31.208, one being the *super-long-flowers* already mentioned, gave approximately the same percentage of *long-flowers* in their progenies (32.213, 32.214, 32.215, table 8). By contrast, 31.209 contained *long-flowers* of only one type, and in the progeny of one of these (31.209P43) the *long-flowers* plants again all appeared to be equivalent (32.217).

Another type which sometimes appears in *long-flowers* populations and is less distinct than the super-long type, has flowers somewhat longer than

normal but not as long as *long-flowers*. Probably the plants in this group do not represent a uniform type for they show considerable variation in habit and flower shape. In two cases, however (32.201 and 32.228), the plants of this type were distinct from the rest of the population and have been classed together as a new type in table 2. In other populations where this type was not distinct, the plants have been recorded as variants.

In its transmission, as well as in its morphological expression, *long-flowers* resembles a trisomic type such as *enlarged* or *large-lax*. It differs from the trisomic type, however, in the considerable percentage of monosomics produced and in the genetic behavior of some plants of its extreme type, *super-long-flowers*.

TABLE 9

Progenies of normal from deformed.

POPULATION	n	f	m	n/s	TOTAL
31.204	46	..	..	2	48
32.202	47	..	..	3	50
32.212	47	2	..	1	50
32.216	50	..	..	..	50
32.220	34	..	15	1	50
Total	224	2	15	7	248

Five populations have been grown from plants classed as *normal* which occurred in *deformed* progenies (table 9). Two of these populations were derived from *normal* plants in immediate progenies of *deformed* (31.204 and 32.220) while a third (32.202) carried one of these *normal* lines (31.204) to the X_5 generation. Of the other two populations, one (32.212) came from a *normal* in a line which had been segregating for *fluted* for three generations, while the last (32.216) came from a *normal* in a line segregating for *long-flowers*. These five populations in contrast to all others from *deformed*, were uniform in habit, growth rate, and morphological features. Their fertility was so much greater than *deformed*, *fluted*, and *long-flowers* that they had completed their blooming period much before other types. Aside from the *mammoth* in 32.220, which have already been discussed, few variants occurred. The occurrence of two *fluted* in 32.212 may be ascribed to the fact that this culture was derived from *fluted*. It is therefore clear that pure breeding races phenotypically equivalent to *control* may be derived from *deformed*. It is probable, however, that even if the same numbers and kinds of genes are present in these plants as are present in the *control*, their structural organization may be quite different, due to transformations which have occurred in their *deformed* ancestry. Thus, these various *normals* from *deformed* might give different genetic results in crosses with *control*, a point which is being determined.

That plants which are close to *control* phenotypically may be genetically quite different was shown in the case of 31.200P8. It was of normal habit and possessed flowers of normal size (52×33 mm) which, however, suggested *fluted* in shape and showed a small amount of *carmine-coral* variegation. An extended series of *fluted* "plus" and "minus" types occurred in 32.200, the selfed progeny of this plant. Aside from 12 plants which did not differ significantly from *normal*, 3 *fluted* and 12 *mammoth* (presumably *fluted-mammoth*) are the only plants in this population which fall readily into known F chromosome types. The remainder of the population consisted of plants differing from *fluted* in greater or lesser degree. Thus 15 plants possessed erect close-ranked leaves typical of *fluted* but had flowers which were close to *normal* in length, rather than shorter as in *fluted*, and differed from *fluted* in flower shape. Six other plants resembled *fluted* in flower shape or size, or in both, but differed from *fluted* in some other morphological feature. One plant was close to *fluted* in habit and in flower shape and size but differed in leaf shape and showed *carmine-coral* variegation together with a small amount of white flecking of the corolla characteristic of *deformed*. One plant was long-flowered with a long petiolate leaf-base. Such a collection of *fluted* variants is a clear demonstration that the characteristic *fluted* morphological complex is being subdivided through production of viable by-products of alteration, the reflection of which has not before been reported.

CYTOLOGICAL EVIDENCE

The interpretation of the genetic results just discussed is based upon the following propositions derived from cytological evidence concerning the chromosomal constitution of *deformed* and its products. (1) The X-ray dosage, applied at prophase to the egg from the fertilization of which with an untreated pollen grain the original *deformed* plant arose, produced an "attachment" between two homologous chromosomes, presumably the two F's. "Attachment" in this sense involves a permanent physical union between chromosomes, with the result that disjunction of the pair involved does not occur at IA (figure 8). (IM, IA, IT = metaphase, anaphase, and telophase of first meiotic division; IIM, IIA, IIT = corresponding stages of the second meiotic division.) In origin "attachment" may be thought of as differing from translocation in the important point that whole chromosomes are "attached," producing a single unit with two spindle-fiber insertions. The original interpretation (GOODSPEED and AVERY, 1930, p. 313) given to this product of attachment was that "opposite ends of a single whole chromosome have become united each to one member of a pair with which the single chromosome is not homologous." While it is possible that chromosome material from some other chromosome than

the F was involved in the transformation leading to the attachment of the F chromosomes, the genetic data at present indicate that only the F chromosomes are concerned. The present interpretation of the cytologically observed attachment and its products involves such a union of F chromosomes as is shown in figure 8, a. The structure and behavior of the attached

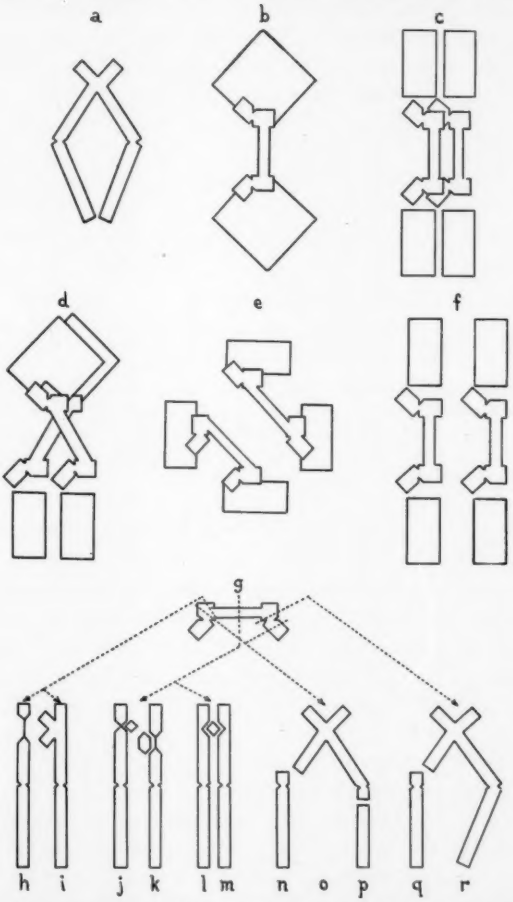


FIGURE 8.—Diagrammatic representation of behavior of attached F chromosomes in meiosis: a, diplotene; b, IIM, following the distribution of parts of the two F chromosomes to opposite poles; c, IIA, showing division of attached F's; d, e, f, IIT, showing three different orientations of the granddaughter nuclei, in each case chromatin bridges connecting two nuclei whose chromosomes were not derived from the same IIM nucleus; g, condition of the attached chromosomes at IIT, the dotted lines showing points of breakage of the chromatin bridge, to give rise to the products h to r.

units corresponds to this interpretation. If the force repelling the two homologous chromosomes at IA is sufficient to produce the chromatin bridge by attenuating the chromatin of the two chromosomes involved, the difference in degree and place of attenuation of the bridge leads to different apparent points of attachment as seen at IT to IIT, and this fact was responsible for the earlier interpretation. While part or all of this earlier interpretation may be valid, we have preferred to use the simpler

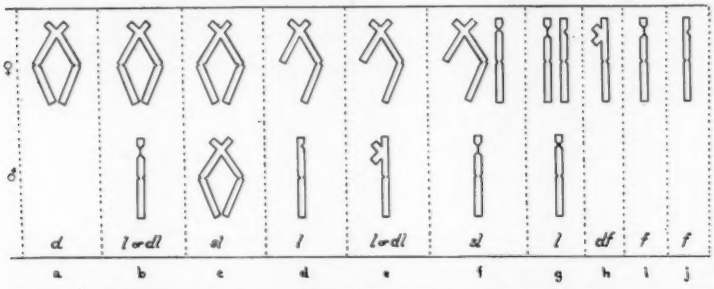


FIGURE 9.—Diagrammatic representation of the F chromosome constitution of some of the gametes of *deformed* and of their union to give the following types which occur in progenies of *deformed*: a, *deformed;* b, *long-flowers* or *deformed-long-flowers;* c, *super-long-flowers;* d, *long-flowers;* e, *long-flowers* or *deformed-long-flowers;* f, *super-long-flowers;* g, *long-flowers;* h, *deformed-fluted;* i and j, *fluted.*

one here described. (2) The chromosome behavior of such a unit is thought of in terms of duality in spindle-fiber insertion and, particularly, so far as meiosis is concerned, of breakages which occur at IIA. During mitosis the relation of this unit to the spindle will, at times, result in its lagging and

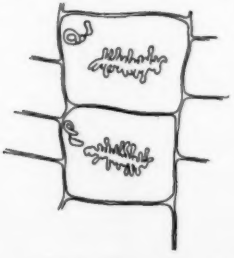


FIGURE 10.—Two adjacent cells from a longitudinal section of a root of *deformed*. In each cell a complex unit is distinct from the metaphase plate. For comment, see text (p. 509). (Paraffin; × 1550.)

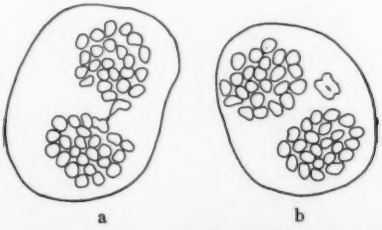


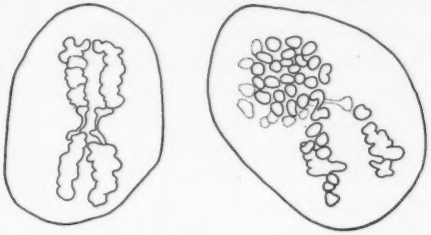
FIGURE 11.—IIM in an X_2 *deformed* plant (29.137P14): a, 24 chromosomes in each plate, the F chromosomes having been distributed to opposite poles at IA, but remaining attached by a chromatin bridge; b, 23 chromosomes in each plate, the attached F chromosomes lagging between the daughter nuclei. (Paraffin; × 1550.)

elimination (figure 10). (3) Products of IIA behavior are normal F's, deficient F's, and F's with various amounts and kinds of additions (figure 8, h–r). (4) Elimination in mitosis of this unit produced by attachment gives rise to abnormalities in tissue production and alignment. Certain of these points are illustrated by the diagrams in figure 8.

The mosaic-like expression of the *deformed* condition is a reflection of somatic elimination of F chromosomes as a result of their attachment. The attached chromosomes may be left in the plasma at telophase of mitoses in *deformed* plants, due to the mechanical difficulties sometimes arising in the separation and distribution of the products of division of the complex unit they constitute. Thus, figure 10 shows two adjacent cells, both in metaphase, from a longitudinal section of a root of a *deformed* plant. Here, in each cell, is a complex double unit, distinct from the metaphase plate, and beginning to degenerate. The fact that these units in adjacent cells appear to be structurally identical, indicates that they are products of delayed division of a complex unit which lagged in the preceding anaphase. The ultimate result of such elimination is the tissue degeneration which can be seen to occur in limited regions of most growing points of *deformed* plants. Nullo-F plants are not known to occur, due presumably to their zygotic lethality, and, similarly, tissue degeneration characteristic of *deformed* probably is a product of the nullo-F condition. The fact that the *deformed* condition has been preserved through five generations on selfing of *deformed* plants (figure 6) indicates that the attached unit, although it is being periodically eliminated in mitosis, is present in most if not all cell lines which are capable of terminating their mitotic cycle in sex-cell formation. Apparently the degrees of expression of the *deformed* condition depend upon the amount of elimination in early growth stages. Thus, the large amount of inviable seed and of death in earlier or later seedling stages which occurs, is a reflection of early and considerable elimination, with the result that not a sufficient amount of normal tissue is available to permit maturity to be attained.

Cytologically, the attached F's present in *deformed* plants are most conspicuous at meiosis because of their abnormal anaphase behavior (figure 8). At IM, the product of attachment may be indistinguishable from other bivalent chromosomes, but at IA the situation involved becomes evident because of the lack of disjunction of the two attached members (figure 11, a). Now, within the normal *tabacum* set, the F chromosome is long with median spindle-fiber insertion (CLAUSEN 1930). The attachment under discussion involves a union of the two F's at a point between the spindle-fiber insertion and one end, as shown in figure 8, a. If attachment was induced at an early meiotic stage, as seems probable, it might represent a permanent union at a point of chiasma formation.

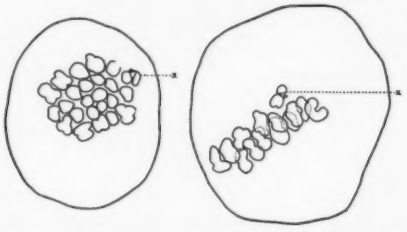
The failure of disjunction of the two attached chromosomes at IA is evidenced by three different types of end product at IT. Most frequently separation of portions of the attached F's takes place, and, along with products of normal distribution, these portions are present in the daughter nuclei. Because of the attachment of the two F's, however, a chromatin



a b

FIGURE 12.—IIA in an X_2 *deformed* plant (29.137P14). The attached chromosomes have divided and form a chromatin bridge between nuclei whose chromosomes were not derived from the same IIM nucleus. Two different orientations of the four granddaughter nuclei are shown in a and b. (Paraffin; × 1550.)

bridge is formed at IT (figure 11, a). In other cases the attached F's fail to reach either pole and lag between the daughter nuclei, where they may be seen as a complex double unit (figure 11, b). At other times, the entire product of attachment goes to one pole, or rarely, the strain caused by the distribution of portions of the two chromosomes to opposite poles may result in the breaking of the attachment at IT. Either of these latter two



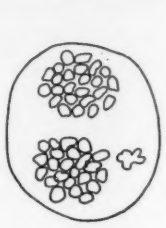
a b

FIGURE 13.—IM in *fluted*, derived from *deformed* (31.207P46): a, IM, polar view with 23_{II} plus the F chromosome ("x"), which is of abnormal shape; b, IM, side view (all chromosomes not shown), F chromosome above the plate ("x"). (Aceto-carmine; × 1140.)

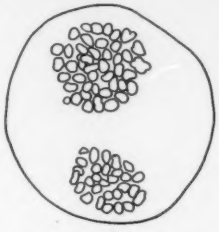
types of behavior give IT and IIM plates which show neither a connection nor a lagging unit. The relative frequencies of these alternative behaviors were determined in the case of an X_2 *deformed* to be 94:56:56, or approximately 2:1:1.

The critical period for the survival of the attached F's in their quanti-

tatively unaltered condition is, however, from IIM to IIT, when the four granddaughter nuclei are becoming most widely separated in the cell. Thus, if the attached units have been distributed to the same pole at IA, they may enter the gamete unaltered. If, on the other hand, they have been distributed to opposite poles at IA, at IIM the connecting chromatin



14



15

FIGURE 14.—IIM in *long-flowers* derive from *deformed* (31.209P33) showing 23 and 24 chromosomes at IIM and the F chromosome with attached segment lying in the plasma. (Aceto-carmine; × 1140.)

FIGURE 15.—IIM in triploid from *deformed* (31.212P19), showing 28 plus a fragment in one plate, and 43 plus a fragment in the other. (Aceto-carmine; × 1140.)

bridge will, itself, divide along with the chromosomes. As the daughter halves separate at IIA, two chromatin bridges will be formed, each connecting two nuclei whose chromosomes were not derived from the same IIM nucleus (figure 8, c; 12). The stresses under which the bridges are placed as the nuclei separate, produce breakage. The various regions of fracture are apparently determined by the relation of the point of attachment to the IIA forces operating in the cell.

a

b

FIGURE 16.—IIT in *long-flowers* (30.115P12), showing chromatin bridges connecting nuclei as in *deformed* (see fig. 12). (Brazilin; ×1140.)

At IIT, the products of breakage are strikingly apparent (see GOODSPEED and AVERY 1930, Plate 19, figures 10–14). Thus, if the break occurs near one spindle-fiber insertion, two units result, one of which is equivalent to half of an F chromosome, and the other to an F chromosome with an additional half attached (figure 8, q, r). If the break occurs near the point of initial attachment, one of the resulting F chromosomes may have a portion deleted, while the other will have this segment added (figure 8,

h, i). It is also possible that a double break may result in the production of a unit consisting of the same two halves of the F chromosome attached (figure 8, n, o, p). As to the other two halves, they are lost or preserved as larger fragments, depending upon whether or not they possess spindle-fiber insertion regions. In addition, very small fragments are produced following the breaking of the bridge (figure 8, j–m). These various products of the action of the IIA and IIT forces upon the chromatin bridges

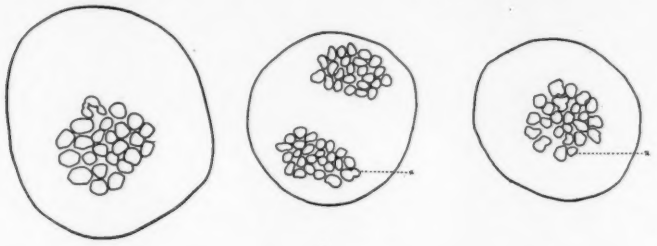


FIGURE 17.—PMC from two shoots of a chimeral plant (32.217P4), showing different chromosome constitutions: a, IM in *long-flowers* shoot, $23_{II}+1_{III}$; b, IIM in *long-flowers* shoot, 24–25; the chromosome marked "x" has an attached segment; c, IM in short-flowered shoot, $23_{II}+1_{I}$, the univalent marked "x." (Aceto-carmine; × 1275.)

correspond closely to the kinds of F chromosomes found in progenies of *deformed* plants. Probably none of these products is structurally equivalent to an F in *control*, and yet genically it may be more nearly equivalent to such a unit. Thus, the genetic evidence shows that the F in *fluted* from *deformed* is genically close to normal F, and yet, as seen at IM in these plants, this unit, conspicuous in the monosomic condition, is not struc-

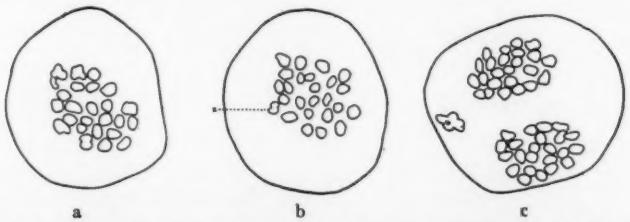


FIGURE 18.—Meiosis in *deformed-long-flowers* (30.115P27): a, IM, $23_{II}+I_{III}$; b, IM, $24_{II}+1_{I}$, the univalent marked "x"; c, IIM, 23 chromosomes in one plate, 24 in the other, and an F with attached segment in the plasma. (Brazilin; × 1140.)

turally equivalent to normal F. Thus, as shown in figure 13, it occurs as two discrete segments physically united at one point. The chromosomes present in certain *long-flowers* derivatives are even more strikingly distinct from normal F, for here there is often a large segment attached to an other-

wise normal chromosome (figure 14). The derivation of such chromosomes from the unit present in *deformed* is shown in figure 8, g–r. In *deformed* plants it has been observed that the connection of IIM plates by means of the attached F's may lead to the formation of "restitution nuclei," and thus of somatic gametes. Two plants from *deformed* have proved to be triploids, 31.212P19 (figure 15) and 32.207P1 (GOODSPEED 1930).

The fact that *deformed*×*control* gives no *deformed*, but gives *long-flowers* and *fluted*, is further evidence of the chromosomal mechanism operating in the production of gametes in *deformed* plants. The non-appearance of *deformed* is due to the contribution by the *control* of a normal F chromosome. If the *deformed* gamete contributes a single F derived from such a breaking of the bridge, as is shown in figure 8, l, m, a *normal* plant is produced. The occurrence of *fluted* in progenies of *deformed*×*control* is the result of the lack of the entire attached unit in the egg. On the other hand, the long-flowered plants in such progenies are, as noted above, triplo-F. Obviously there is a possibility that such a plant contains three unattached F's as a result of the inclusion of both products of the break at IIM in the production of the egg (figure 9, g). If the *deformed* egg contains attached F's, the addition of a normal F from *control* determines lack of deformity and gives *long-flowers* (figure 9, d). Thus, in this latter case, although the attached F's are undoubtedly present and being eliminated in mitosis as in *deformed*, the unattached F prevents the degeneration of the cells involved and thus the external evidence of tissue deformation (figures 16, a and b).

That chromosome elimination takes place in somatic tissues of *long-flowers*, was made evident in the progenies from *deformed*×*coral*, where the structurally unaltered F contained the recessive flower color factor, while the dominant carmine factor was introduced with the F chromosome complex from *deformed*. In these progenies, the elimination of the F chromosome from the gametes of *deformed* could be measured by the number of *coral-fluted* plants, and the elimination of F chromosomes derived from *deformed* in somatic mitoses could be followed by the occurrence of *carmine-coral* variegation (figure 7, g). This kind of variegation occurred in *long-flowers*, as well as in plants with flowers of normal length, and is an indication that the attached chromosomes present, responsible for *long-flowers*, are occasionally eliminated in somatic divisions.

Further evidence that chromosome elimination takes place in *long-flowers* without producing deformity, is found in the fact that three different plants of this phenotype have given rise to shoots from the base of the central stalk on which all the flowers had lost the *long-flowers* character, due to the loss of one or more F's from the cells of their central axes giving rise to these shoots. In the case of 32.217P4 the loss could be ob-

served cytologically by comparison of the PMC produced by the central axis and the side shoot. Thus, $23_{II}+1_{III}$ at IM (figure 17, a) and 24–25 at IIM (figure 17, b) were the characteristic configurations in PMC from the *long-flowers* axis, while the PMC from the short-flowered shoot showed only $23_{II}+1_{I}$ (figure 17, c).

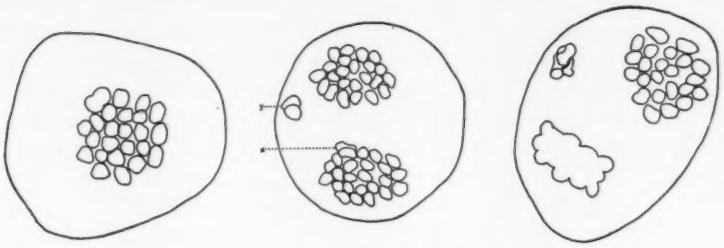


FIGURE 19.—Meiosis in *super-long-flowers* (32.209P39): a, IM, $23_{II}+1_{IV}$; b, IIM, 25 chromosomes in one plate, two of which were not disjoined at IA ("x"), 23 in the other plate, and two products of non-disjunction in the plasma ("y"); c, IIM, 23 plus a quadrivalent in the plasma. (Aceto-carmine; × 1230.)

The genetic evidence given in table 3 shows that non-*deformed* lines of *fluted*, *long-flowers* and *mammoth* have been derived from the original *deformed* plant. The origin of such lines is as follows. Where the attached unit is entirely absent in a gamete, due to elimination in meiosis, the gamete being therefore nullo-F, its union with a haplo-F gamete, produced through

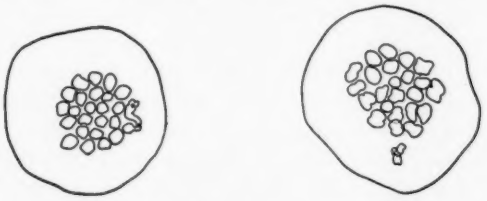


FIGURE 20.—IM in *super-long-flowers* (31.208P50), showing 23_{II} plus a quadrivalent involving a "chain" of chromosomes. (Aceto-carmine; × 1140.)

FIGURE 21.—IM in *fluted-mammoth* (31.212P32); $23_{II}+1_{I}$, the chromosome in the plasma showing a type of structural abnormality of the F chromosome. (Aceto-carmine; × 1140.)

breaking of the attachment, gives *fluted* (figure 9, i and j). On the other hand, *long-flowers* represents essentially the triplo-F condition. It may be a product of the union of a gamete containing the attached unit and a haplo-F gamete derived as in the case of *fluted* mentioned above (figure 9, b). At IM such a plant will show either 24 or 25 units. In the former case one unit is structurally equivalent to a trivalent, two members of which

are attached (figure 18, a); in the latter there is a univalent not associated with the attached unit (figure 18, b). When the attachment is broken to give two normal F's, their union with a normal gamete will also give *long-flowers* (figure 9, g). Again, when the breaking of the attachment is such that a segment of an F is still attached to the other F, so that the segment lacks a spindle-fiber insertion, a gamete results which, united with a gamete containing a normal F, gives *long-flowers* (figure 9, d). A plant so derived gives evidence of its chromosome constitution at IIM, where the two plates contain respectively 23 and 24 chromosomes with a unit in the plasma, the latter showing an F with a segment attached (figure 18, c). As already noted, the progenies of such plants in succeeding generations consist of *normal*, *long-flowers*, and *fluted*. This result depends upon the kinds of gametes produced by the mechanism just described. *Super-long-flowers* occur in progenies of *deformed* and particularly in derived *long-flowers* lines. They are found to represent the tetra-F condition resulting from union of gametes containing two attached F's or an F with an attached segment, or two detached F's (figures 9, c, f; 19, and 20).

The appearance of *mammoth* in this complicated situation is explicable, since the F complex is concerned in the spontaneous appearance of this type (CLAUSEN 1931). Its genetic behavior is that of a monogenic recessive, but it is possibly a product of structural chromosome alteration (DARLINGTON 1932, p. 242). In untreated lines, *fluted-mammoth* and *long-flowers-mammoth* have been derived from *mammoth* as a result of the occasional production of haplo- and triplo-F types associated with the presence of the alteration which gives *mammoth*. On the other hand, the occurrence in X-ray derivatives of *deformed-mammoth*, *deformed-fluted-mammoth*, and *deformed-long-flowers-mammoth*, calls for further comment. Their occurrence is involved in the following considerations, which also apply to the occurrence of *deformed-fluted* and *deformed-long-flowers*.

All of these types which have the *deformed* character in common probably represent products of breakages of the attachment occurring in the *deformed* parents (figure 9). Of course, *deformed-mammoth* and *deformed-long-flowers-mammoth* might be related to the transmission of the two F's in their attached condition, if the alteration responsible for the production of the *mammoth* condition had occurred in all the F's, or in all except those which are deficient for the non-*mammoth* allelomorph. It seems more probable, however, that the *deformed* condition in most of these particular derivative combinations is due to the presence of unattached F's whose structural alterations are such as to induce somatic elimination. For example, 32.207, the progeny of a *deformed*, possessed a large number of *deformed-fluted* (figure 6), a phenotype which had occurred in the case of only one plant previously. Apparently, here, the attached F's seen in the

parent (31.205P35) must have been detached to give F's genetically close to normal but structurally so modified that their reproduction in somatic mitoses is impaired and leads to their occasional elimination from certain cells, which are therefore nullo-F and inviable. *Deformed-long-flowers* do not show the extent of tissue abnormality which is characteristic of *deformed-fluted*, and this indicates that the complete elimination of the F's is not as frequently accomplished in the former case. In such plants two units involving the F attachment complex must be sufficiently abnormal in structure to induce their elimination (figure 9, e). One of these units is in some cases the F with a segment attached, as already described and shown in figures 14, and 18, c, where the attached segment is responsible for the *long-flowers* type. The other F in this case may be equivalent to

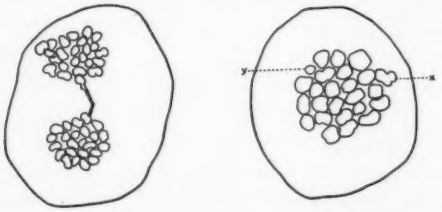


FIGURE 22.—Chromosome attachment, and one of its products: a, IIM in a plant showing *carmine-coral* variegation (31.200P8), 24 chromosomes in each plate, one of which is attached to a chromosome in the other plate; b, IM in *fluted* variant (32.200P46), derived from 31.200P8, 23_{II}, an F chromosome with a segment attached ("x"), and a fragment ("y"). (a, Aceto-carmine; × 1140. b, Aceto-carmine; × 1100.)

that in *deformed-fluted*. In the case of *deformed-mammoth*, therefore, there may be two structurally altered but unattached F's by which the *mammoth* character is transmitted. The modified F present in a *fluted-mammoth* is shown in figure 21. In what way the structural modification that is evident in this case is related to the production of the *mammoth* character is not clear from the evidence available. It is clear, however, that the F chromosome shows structural alterations in all types in which the *deformed* condition is expressed morphologically.

Carmine-coral variegation may be related to chromosome attachment, as is shown in figure 22, a. Here the chromatin bridge connecting IIM plates is seen in a plant of this type. The chromosome constitution of a *fluted* variant derived from this plant is shown in figure 22, b. Here, in addition to 23_{II}, there is a fragment and a univalent F chromosome which has an attached segment, both derived from the breaking of the chromatin bridge in the parent plant.

Of the many products of attached F's observed, the structurally modi-

fied F chromosome present in an X_3 *fluted* deserves mention. At IM in PMC of this plant, the unpaired F chromosome could be seen among the 23_{II} as a lightly-staining, deeply constricted unit somewhat larger than the normal F. Further study indicates that it is an F with a small attached segment. Such an F frequently lags at IA, and divides in the plasma at IIM. At IIT the products of this equational division are very clear because of their isolation in the cell (figure 23). It was sometimes found that in dividing, one daughter-half of the attached segment became detached (figure 23, a), so that at IIT there was one daughter-F with the attached segment and another without the attached segment, the latter being free

FIGURE 23.—IIT in *fluted* (30.113P14), showing division of the F chromosome with a segment attached, to give one daughter F with the attached segment, one without an attachment, and the detached segment free in the plasma. (Aceto-carmine; × 1140.)

in the plasma (figure 23, b, c). This loss of the attached segment in a division corresponding to a somatic one suggests that a similar process may be operating in the production of "mottled" Drosophila described by PATTERSON (1932a). He has shown that "mottled" is the product of an unstable translocation, and has postulated that "it is possible for the chromosome with the attached fragment to split in such a manner that one cell will receive a daughter chromosome bearing the undivided fragment, while the sister cell will receive the other daughter chromosome without the fragment." The mechanism here illustrated, that is, the loss of one product of division of the fragment, would account equally well for PATTERSON'S results.

DISCUSSION

The evidence, genetic and cytological, concerning the "*deformed* assemblage" which has been presented above demonstrates the far reaching consequences of chromosome attachment of the type obtaining in this instance. The attachment concerned here has been productive of (1) races transmitting quantitative chromosomal variations (the monosomic, trisomic and tetrasomic conditions); (2) pure-breeding races transmitting recessive character contrasts which may be, however, products of structural chromosome alteration (*coral* and *mammoth* derivatives); (3) "eversport-

ing" races, products of mosaicism (*deformed*), transmitting the "ever-sporting" tendency.

We have shown that the *deformed* condition is a reflection of somatic elimination of F chromosomes as a result of a physical attachment in which they are involved. In addition to confirmatory cytological evidence, the presence of equal numbers of monosomic and trisomic types in progenies of *deformed* indicates the behavior in meiosis of this product of attachment. The genetic behavior of other elements of the *deformed* assemblage depends upon the initial presence of the attachment together with products of its breakage. These products may be, structurally, more simple and give relative simplicity in genetic behavior, as in *fluted* races derived from *deformed*. They may, however, be complex as is evidenced by the behavior of derived *long-flowers* lines, for example.

Those instances in which the genetic behavior of a *deformed* derivative cannot readily be related to products of the attachment which determined its phenotype, are interpretable in terms of secondary structural alteration or of elimination of products of the initial chromosome reorganization involved. Thus, the F chromosomes in *deformed-fluted* (30.113P15) which gave all *mammoth* suffered alteration in mitosis, while the added F responsible for the production of *deformed-long-flowers* (30.115P31) was eliminated in somatogenesis with the result that the progeny contained no *long-flowers*.

The capacity of high frequency radiation to induce quantitative chromosomal alterations has been recognized for some time. One of the first chromosomal variants thus induced, the cytogenetics of which was fully reported, was *Nubbin*, a compound chromosome derivative in Datura (BLAKESLEE 1927). It gave five morphologically distinct, variant types, one of which was *Nubbin*, in whose progeny the same complex segregation occurred. Thus, in genetic behavior *Nubbin* suggests *deformed*. However, all five of the variant types produced by *Nubbin* are trisomics, two being primary trisomics, two tertiary trisomics, and one *Nubbin* like the parent. *Deformed*, on the other hand, is not itself a trisomic, and its derivatives in addition to *deformed*, are monosomic, trisomic and pure-breeding recessive disomic for the F chromosome.

As already noted, *deformed* is comparable to STEIN'S (1926) "farb- und-formdefekt" strain of Antirrhinum in external morphology and in the nature of the tissue degeneration involved. In particular her "Phytocarcinome" (PCa) R1053 (STEIN 1930), induced by radium treatment of an embryo, is of interest here. (STEIN designates her radium treated generation "R," and the progeny derived from it "R_1," while we use R_1 and R_2 respectively.) The derivatives of R1053 are reminiscent of those from *deformed* so far as the complexity of types and their interrelations are con-

cerned. STEIN (1932) has shown that her case may behave as a Mendelian recessive, breeding true and segregating in F_2 in crosses with normal. She, also, finds evidence of mitotic and meiotic abnormalities which she interprets as induced by the tissue degeneration produced by a recessive gene. On the other hand, we have shown that *deformed* is not the product of an induced recessive gene mutation and, further, that the mitotic and meiotic abnormalities it shows are the cause and not the effect of tissue degeneration.

The manner in which lethal deficiency effects operate in the production of the *deformed* condition calls for comment. PATTERSON (1932b) has shown that the loss from one of the X chromosomes in the zygote of Drosophila, of a small "viability region," possibly a single gene, has the effect of a dominant lethal. When lost from a cell during somatogenesis the cell descendants, in combination with non-deficient cells, give somatic tissues exhibiting mosaicism. In *deformed*, something of the nature of such a "viability region" is concerned but here it appears to have a recessive, in place of a dominant, lethal effect. In this connection, it is to be noted that in STEIN'S PCa individuals and, also, in the case of *deformed*, the lethal effect apparently is not strictly limited to cells deficient for the "viability region" but extends its influence to neighboring cells.

Doubtless many cases involving the occurrence of gynandromorphs, mosaics, mottles, and variegated or "eversporting" individuals are related to somatic elimination of chromosomes which have become structurally abnormal. In Drosophila, L. V. MORGAN (1929) found a line possessing an abnormally shaped X (the "U" line). It gave a high percentage of gynandromorphs in which it could be shown that the abnormal X was eliminated in mitosis. This chromosome was originally one of the two attached X's and doubtless its structural abnormality was attained during detachment. In this regard it is comparable to certain abnormal F chromosomes in derivatives of *deformed*.

In many features the mottled-eyed Drosophila case as reported by PATTERSON (1932a) resembles that of *deformed*. Both are products of X-ray treatment and in certain respects are comparable as to the morphological expression involved, genetic behavior, and, also, in respect to cytological interpretations offered. The occurrence among mottled-notched females of "individuals that show every degree of mottled and notched conditions" is paralleled by the variability of expression of the *deformed* condition and of *carmine-coral* variegation. Again, hyperploid *long-flowers* derivatives of *deformed* produce *deformed* offspring, and similarly PATTERSON reports that "fertile hyperploid flies really constitute a mottled line, from which a stock can be established." Finally, the interpretation of both cases involves the elimination of chromosome material during mitosis. On

the other hand, mottled involved an "unstable translocation"—a small segment of the X chromosome translocated to the fourth chromosome. In *deformed*, however, more than a whole chromosome is subject to elimination. It may vary in its constitution as it occurs in different individuals and in succeeding generations of *deformed*, but so long as it is subject to loss, this chromosome is seen to differ morphologically and structurally from its condition in *control*. (MULLER [1930] has noted that all cases of mottled involve some type of gene rearrangement—deletion, translocation, or inversion.)

Attention has been called above to certain reported instances in which treatment with high frequency radiation has produced chromosomal variants exhibiting complex interrelationships, tissue degeneration which is hereditary, and mosaicism involving chromosome elimination. The data submitted here as to the cytogenetics of the "*deformed* assemblage" indicate that a unique situation obtains in this case since all of these various phenomena are concerned and are capable of a consistent genetic and cytological interpretation on the basis of chromosome attachment and resulting elimination.

LITERATURE CITED

BLAKESLEE, A. F., 1927 *Nubbin*, a compound chromosomal type in Datura. Ann. New York Acad. Sci. **30**: 1–29.

CLAUSEN, R. E., 1930 Inheritance in *Nicotiana tabacum*. X. Carmine-coral variegation. Cytologia **1**: 358–368.

1931 Inheritance in *Nicotiana tabacum*. XI. The fluted assemblage. Amer. Nat. **65**: 316–331.

CLAUSEN, R. E. and GOODSPEED, T. H., 1924 Inheritance in *Nicotiana tabacum*. IV. The trisomic character, enlarged. Genetics **9**: 181–197.

1926 Inheritance in *Nicotiana tabacum*. VII. The monosomic character, fluted. Univ. California Pub. Bot. **11**: 61–82.

DARLINGTON, C. D., 1932 Recent advances in cytology. P. Blakiston's Son & Co.

GOODSPEED, T. H., 1929 The effects of X-rays and radium on species of the genus Nicotiana. J. Hered. **20**: 243–259.

1930a Inheritance in *Nicotiana tabacum*. IX. Mutations following treatment with X-rays and radium. Univ. California Pub. Bot. **11**: 285–298.

1930b Occurrence of triploid and tetraploid individuals in X-ray progenies of *Nicotiana tabacum*. Univ. California Pub. Bot. **11**: 299–308.

1931 Die Bedeutung von quantitativen Chromosomenveränderungen. Die Naturwiss. **23**: 476–483.

1932 Cytogenetic consequences of treatment of Nicotiana species with X-rays and radium. Svensk. bot. Tidskr. **26**: 147–162.

GOODSPEED, T. H. and AVERY, P., 1930 Nature and significance of structural chromosome alterations induced by X-rays and radium. Cytologia **1**: 308–327.

MOHR, O. L., 1932 Genetical and cytological proof of somatic elimination of the fourth chromosome in *Drosophila melanogaster*. Genetics **17**: 60–80.

MORGAN, L. V., 1929 Composites of *Drosophila melanogaster*. Pub. Carnegie Instn. Washington **399**: 223–294.

MULLER, H. J., 1930 Types of visible variations induced by X-rays in Drosophila. J. Genet. **22**: 299–334.

PATTERSON, J. T., 1932a A new type of mottled-eyed Drosophila due to an unstable translocation. Genetics **17**: 38–59.

1932b A gene for viability in the X chromosome of Drosophila. Z. I. A. V. **60**: 125–136.

STEERE, W. C., 1931 A new and rapid method for making permanent aceto-carmin smears. Stain Tech. **6**: 107–111.

STEIN, E., 1926 Untersuchungen über die Radiomorphosen von Antirrhinum. Z. I. A. V. **42**: 1–87.

1930 Weitere Mitteilung über die durch Radiumbestrahlung induzierten Gewebe-Entartungen in Antirrhinum (Phytocarcinome) und ihr erbliches Verhalten. (Somatische Induction und Erblichkeit.) Biol. Zbl. **50**: 129–158.

1932 Zur Entstehung und Vererbung der durch Radiumbestrahlung erzeugten Phytocarcinome. Z. I. A. V. **62**: 1–13.

WEBBER, J. M., 1929 A smear method for the study of chromosomes in microsporogenesis. Univ. California Pub. Bot. **14**: 345–352.

BIONOMIC STUDIES ON CERTAIN TELEOSTS (POECILIINAE). III. HEREDITARY BEHAVIOR OF THE COLOR CHARACTER, GOLD

A. W. BELLAMY

University of California, Los Angeles, California

Received February 7, 1933

In an earlier paper (BELLAMY 1928), reference was made to a "golden" variety of *Platypoecilus maculatus* GÜNTH. This report presents data on the hereditary behavior of this character and discusses its relation to the characters previously studied and to the work of FRASER and GORDON (1929) on the same variety. The data, all of which, except for a few matings summarized in table 1, are presented in pedigree form, table 6. They were obtained, in part, in the Hull Zoological Laboratories at the UNIVERSITY OF CHICAGO. Through 1926–1929, work progressed slowly in an improvised laboratory at my home. In 1929 facilities became available for resuming work on the new campus of the UNIVERSITY OF CALIFORNIA at Los Angeles. After many interruptions it has been possible to conclude certain analyses, results of which I wished to study before submitting the results obtained in 1923–1924.

GORDON (1927, 1931) and FRASER and GORDON (1929), have furnished excellent descriptions of the variety known as Gold. It is described as a clear transparent yellow, slightly tinged with pale orange, eyes greenish blue, dorsal fin flushed with red. GORDON finds that the color of the body compares favorably with Deep Colonial Buff, Ridgway Plate 30; dorsal fin of male, Scarlet; and dorsal fin of the female, La France Pink, Ridgway Plate 1. Eyes are Turquoise Green, Ridgway Plate 7. GORDON (1931 pp. 749, 750) notes further that this variety is without spots (macro-melanophores) and stipples (micro-melanophores) with the exception of occasional micro-melanophores in the mid-dorsal region and along the caudal peduncle. The red of the dorsal fin, in my material, often extends down onto the body, occasionally reaching the mid-ventral line near the anal and pelvic fins. In extreme cases erythrophores are present along the caudal peduncle. In the presence of the gene, or genes, for Blue (See GORDON 1931, p. 763 for description) and where the guanophores cover an extensive area the Gold variety takes on a decided silvery cast, which under certain lighting conditions, may require the use of a microscope to effect certain distinction of a few individuals from typical Blue stippled specimens.

Other characters of Platypoecilus, the hereditary behavior of which has been described, fall into what has been interpreted as a sex-linked multiple allelomorphic series. GORDON (1926, 1927) and FRASER and GORDON (1929)

state, however, that the type described as White (BELLAMY 1928) or stipples (GORDON) is not sex-linked but autosomal. This conclusion is natural enough since the micro-melanophore pattern behaves as a simple Mendelian dominant to Gold which is certainly autosomal. As far as one can judge from the available information our differences in opinion or usage appear to be largely rhetorical, since the hereditary behavior of the stipple pattern is readily and consistently described by considering it a wild-type as is done in Drosophila. Geneticists find no particular difficulty in regarding red eye color in Drosophila as either autosomal or heterosomal, depending upon the particular allelomorph under consideration.

In describing genotypes the following symbols are used:

1. $+$ = Wild-type (White, Stipples). The same symbol, $+$, is also used for the wild-type allelomorph of *N*, *P*, and *R*.
2. *g* = Gold.
3. *N* = Nigra. (Stippled. Macro-melanophores arranged in a definite pattern.)
4. *R* = Rubra. (Stippled, red, spotted. While females are usually, but not always, much less red than males, they are more variable and develop the red color later.)
5. *P* = Pulchra. (Stippled, spotted, non-red.)

A Nigra-Gold specimen is non-stippled and shows typical effects of the gene *g*, except in those regions of the body that are obscured by the Nigra pattern. Pulchra-Gold is non-red, non-stippled, spotted, but otherwise showing typical effects of *g*. Rubra-Gold is red, spotted, non-stippled. Separation of Rubra-Gold from Rubra is most easily effected by isolating all Gold young soon after birth, from matings where both phenotypes are expected. The separation can be made in adult fish but it is necessary to use a microscope on many specimens to determine the presence or absence of micro-melanophores.

It will be convenient to use any of the above symbols, except *g* in place of the X (or Z) chromosome symbol, and *O* for the Y (or W) chromosome symbol. Heterosomic or sex-linked characters (genes) are written first. Thus, *RR gg* and *RO gg* represent Rubra-Gold male and female respectively. $++gg$ represents a Gold male; $+Og+$, a female heterozygous for the wild-type allelomorph of Gold and with the heterosomal wild-type allelomorph of the multiple allelomorphs, *R*, *N*, or *P*. In this manner the necessity for giving the wild-type symbol a special subscript label is avoided.

The pedigree, table 6, is so arranged that the reader may compare the data with any hypothesis. Since original record numbers are used it is really a continuation of the pedigree previously published (BELLAMY 1928,

table 3). The type of mating is indicated in the first column where the genotype of the male parent is written first. The second column gives the original record number; third and fourth columns the source of the parents. Where the source of material is unknown, usually obtained from a local dealer, the fact is indicated by the letter *U*. In a few cases material has been taken from unpedigreed stocks kept for varying lengths of time in the laboratory. The number of young born is recorded in the fifth column and in the sixth to twelfth columns the phenotype of offspring. Males are recorded above and females in the line below as indicated for the first mating of the table. Where the color of young has been recorded soon after birth the number is recorded in parentheses under the phenotype they most closely resemble.

Crosses of Gold with the Wild-type were made reciprocally with substantially identical results, (table 1).

TABLE 1

	Gold *gg* × Wild-type ++				
	Wild-type *g*+				
	♂		♀		
F_1	110		129		(Matings 303, 322, 462, 465)
Wild-type ++ and *g*+		Gold *gg*			
♂	♀	♂	♀		
124	144	37	25		(Matings 303.1, 322.1, 450.2, 462.1, 463.1)

Aside from the relatively small number of F_2 Golds, which is probably insignificant with such small numbers, the chief matter for comment is the appearance of F_1 and F_2 Wild-types. They approximate the Wild-type in general appearance, but dominance is not complete except as regards stippling. The dorsal fin is distinctly tinged with red, most marked near the base of the fin, and in many specimens, mostly males, there is a trace of red color on the abdominal wall near the anal and pelvic fins. The red color in the dorsal fin is more conspicuous in males than in females. This tendency toward an intermediate condition indicates that Gold is not simply a fish without stipples but that it has certain positive attributes of its own.

Since *P*, *N*, and *R* may be regarded as belonging to the same allelomorphic series, crosses involving these three patterns and Gold are tabulated together. The record numbers of the matings included in the tabulation, table 2, are given and the reader may study each cross separately by referring to the pedigree, table 6. The letter *C* is used in tables 2 and 4 to replace *P*, or *R*, or *N*.

Separation of Rubra-Gold from Rubra was not considered accurate enough in mating 304.1, the first in which these types appeared, to justify inclusion in the tabulation. Results of this mating are included, however, in table 3.

TABLE 2

	Gold ♂ ++*gg* × Colored ♀ *CO*++								
	Colored ♂♂ *C*+*g*+		Wild-type ♀♀ +*O*g+						
F_1	48		54						(Matings 304, 305, 306)
	Colored		Wild-type		Colored-Gold		Gold		
	♂♂	♀♀	♂♂	♀♀	♂♂	♀♀	♂♂	♀♀	
F_2	65	70	63	86	9	9	14	23	(Matings 305.1, 305.2, 306.1, 306.2)

TABLE 3

	RED				NON-RED			
	SPOTTED		NON-SPOTTED		SPOTTED		NON-SPOTTED	
	♂♂	♀♀	♂♂	♀♀	♂♂	♀♀	♂♂	♀♀
F_1	48	0	0	0	0	0	0	54
F_2	91	106	0	0	0	0	91	119

The results given in table 2 may be compared, to some extent, with the data of FRASER and GORDON (1929, table 2) by reclassifying the data and using their descriptive names (table 3).

Data from reciprocal crosses, Colored male × Gold female, are presented in table 4, and compared with similar data of FRASER and GORDON (1929, table 1) in table 5.

TABLE 4

	Colored ♂ *CC*++ × Gold ♀ ++*gg*								
	Colored				Wild-type (inter-sex ?)				
	♂♂ *C*+*g*+		♀♀ *CO*g+						
	89		54		1				(Matings 317, 388, 389, 390)
	Colored		Wild-type		Colored-Gold		Gold		
	♂♂	♀♀	♂♂	♀♀	♂♂	♀♀	♂♂	♀♀	
	96	57	0	49	27	17	0	17	(Matings 388.1, 388.2, 389.1, 390.1)

TABLE 5

	RED				NON-RED			
	SPOTTED		NON-SPOTTED		SPOTTED		NON-SPOTTED	
	♂♂	♀♀	♂♂	♀♀	♂♂	♀♀	♂♂	♀♀
F_1	89	54	0	0	0	0	0	1 0
F_2	123	74	0	0	0	0	0	66

It will be noted that only matings 304, 304.1, 317, 389, and 389.1 are directly comparable with the results of FRASER and GORDON. The other types of matings, however, involving *N* and *P* are better suited to the analysis in question since, with one exception, all of the phenotypes are easily distinguished and many of the genotypes can be determined by inspection. The distinction between Rubra-Gold and Rubra is mentioned on page 523. By isolating all of the Gold young in mating 389.1 soon after birth the separation of the two phenotypes was easily effected.

Unfortunately a detailed comparison between these data and those of FRASER and GORDON (1929) is not possible, since apparently they have thrown certain phenotypes together. In the headings to their tables 1, 2, 3, and 4, appears the phrase "Gold—(non-spotted, non-red)" and while it is legitimate to infer that the offspring labeled in the tables as "non-red, non-spotted" are Gold, it seems probable that they have simply suppressed certain details for the sake of brevity or to emphasize a particular point. In all of the F_2 matings in tables 2 and 4 above and presumably in FRASER and GORDON'S tables 1 and 2, among others, all colored spotted animals fall into two phenotypes, namely, Gold and not Gold. Likewise the non-spotted F_2 offspring are of two types, Gold and not Gold.

As far as most of my own data go, *R*, *N*, and *P* are satisfactorily treated as "unit characters" depending upon a difference in one pair of genes. The appearance of a non-spotted red male and an "exceptional" red, spotted female in FRASER and GORDON'S laboratory makes it necessary to re-examine the question of the genetic constitution not only of Rubra but also of Nigra and Pulchra. Discussion of this matter follows in the fourth report of this series (BELLAMY 1933) on linkage and non-disjunction in Platypoecilus.

SUMMARY

1. The hereditary behavior of Gold, an autosomal character, in relation to the multiple allelomorphic series, *N*, *P*, *R*, and the Wild-type, are described and compared with FRASER and GORDON'S (1929) crosses between Rubra and Gold.

2. Gold behaves as a simple Mendelian recessive to the Wild-type. Dominance of the Wild-type is not complete.

LITERATURE CITED

BELLAMY, A. W., 1924 Bionomic studies on certain teleosts (Poeciliinae). Genetics 9: 513–529.

1928 Bionomic studies on certain teleosts (Poeciliinae). Color pattern inheritance and sex in *Platypoecilus maculatus* Günth. Genetics 13: 226–232.

1933 Bionomic studies, et cetera. IV. Crossing over and non-disjunction in *Platypoecilus maculatus* Günth. Genetics 18: 531–534.

FRASER, A. C. and GORDON, MYRON, 1929 The genetics of Platypoecilus. II. The linkage of two sex-linked characters. Genetics 14: 160–179.

GORDON, MYRON, 1927 The genetics of a viviparous top-minnow Platypoecilus; the inheritance of two kinds of melanophores. Genetics 12: 253–283.

1931 Morphology of the heritable color patterns in the Mexican killifish, Platypoecilus. Amer. J. Cancer 15: 732–787.

TABLE 6

MATING	RECORD NUMBER	SOURCE OF MALE	SOURCE OF FEMALE	YOUNG BORN	GOLD	WILD-TYPE	NIGRA	NIGRA-GOLD	RUBRA	RUBRA-GOLD	PULCHRA	PULCHRA-GOLD	SEX NOT RECORDED
gg×*gg*	302	*U*	*U*	71	♂29	..	..	..	..	..	..	..	12
					♀30	..	..	..	..	..	..	..	
gg×*gg*	302.1	302	302	136	29	..	..	..	..	..	..	..	77
					30	..	..	..	..	..	..	..	
gg×++	303	302	260	98	..	38	..	..	..	..	..	..	38
					..	22	..	..	..	..	..	..	
g+×*g*+	303.1	303_1	303	177	(47)	(130)							
					10	23	..	..	..	..	..	..	120
					2	22	..	..	..	..	..	..	
++*gg*×*RO*++	304	302_1	44.3.2	102	..	..	..	..	11	..	..	..	82
					..	9	..	..	..	..	..	..	
R+*g*+×+*Og*+	304.1	304	304	147	(31)	(101)							
					3	14	..	..	17	..	..	..	62
					4	20	..	..	27	..	..	..	
++*gg*×*NO*++	305	302_1	217.2	70	..	..	23	..	..	..	..	..	25
					..	22	..	..	..	..	..	..	
N+*g*+×+*Og*+	305.1	305	305	49	(14)	(35)							
					0	1	5	0	..	..	..	..	31
					4	1	5	2	..	..	..	..	
N+*g*+×+*Og*+	305.2	305	305	140	(27)	(100)							
					7	16	20	4	..	..	..	..	56
					2	21	12	2	..	..	..	..	
++*gg*×*PO*++	306	302_1	83.1.2	40	..	..	..	..	..	..	20	..	3
					..	17	..	..	..	..	..	..	

TABLE 6 (Continued)

MATING	RECORD NUMBER	SOURCE OF MALE	SOURCE OF FEMALE	YOUNG BORN	GOLD	WILD-TYPE	NIGRA	NIGRA-GOLD	RUBRA	RUBRA-GOLD	PULCHRA	PULCHRA-GOLD	SEX NOT RECORDED
P+*g*+×+*Og*+	306.1	306	306	228	(41)	(187)							
					6	26	..	..	..	..	27	4	78
					5	40	..	..	..	..	39	3	
P+*g*+×+*Og*+	306.2	306	306	95	(10)	(73)							
					2	20	..	..	..	..	13	1	17
					2	24	..	..	..	..	14	2	
gg×*gg*	311	*U*	*U*	67	25	..	..	..	..	..	..	..	28
					14	..	..	..	..	..	..	..	
RR++×+*Ogg*	317	44.2.1	311	157	..	1	..	..	40	..	..	..	69
					..	..	..	..	47	..	..	..	
++×*gg*	322	117.1.1	302	93	..	25	..	..	..	..	..	..	33
					..	35	..	..	..	..	..	..	
g+×*g*+	322.1	322	322	56	(17)	(39)							
					13	18	..	..	..	..	..	..	3
					2	20	..	..	..	..	..	..	
P+++×+*Ogg*	324	263	311	66	..	4	..	..	..	..	12	..	28
					..	9	..	..	..	..	13	..	
N+++×+*Ogg*	325	265	311	6	..	1	1	..	..	..	..	..	2
					..	1	1	..	..	..	..	..	
++*gg*×*NOgg*	357	302	305.1	13	..	..	..	8	..	..	..	..	0
					5	..	..	..	..	..	..	..	
N+++×+*Ogg*	358	305.1	311	37	..	11	5	..	..	..	..	..	6
					..	8	7						
N+*g*+×+*Ogg*	359	305.1	311	14	1	6	0	1	..	..	..	..	0
					0	5	1	0	..	..	..	..	

TABLE 6 (Continued)

MATING	RECORD NUMBER	SOURCE OF MALE	SOURCE OF FEMALE	YOUNG BORN	GOLD	WILD-TYPE	NIGRA	NIGRA-GOLD	RUBRA	RUBRA-GOLD	PULCHRA	PULCHRA-GOLD	SEX NOT RECORDED
R+g+×+O++	360	304	314	70	..	7	..	..	11	..	..	..	25
					..	9	..	..	18	..	..	..	
R+g+×+Ogg	361	304	302	59	(22)	(37)							
					3	7	..	..	6	4	..	..	15
					5	9	..	..	8	2	..	..	
PP++×+Ogg	388	Stock	Stock	60	..	..	..	..	..	..	19	..	17
					..	..	..	..	..	..	24	..	
P+g+×POg+	388.1	388	388	47	(10)	(37)							
					..	..	..	..	..	..	14	2	10
					2	7	..	..	..	..	9	3	
P+g+×POg+	388.2	388	388	104	(25)	(79)							
					..	..	..	..	..	..	32	10	19
					5	14	..	..	..	..	18	6	
RR++×+Ogg	389	Stock	Stock	38	..	..	..	..	8	..	..	..	23
					..	..	..	..	7	..	..	..	
R+g+×ROg+	389.1	389	389	73	(16)	(57)							
					..	..	..	..	17	4	..	..	26
					3	10	..	..	10	3	..	..	
NN++×+Ogg	390	Stock	Stock	52	..	..	22	..	..	..	..	..	8
					..	..	22	..	..	..	..	..	
N+g+×NOg+	390.1	390	390	114	(29)	(85)							
					..	..	33	11	..	..	..	..	20
					7	18	20	5	..	..	..	..	

BIONOMIC STUDIES ON CERTAIN TELEOSTS (POECILIINAE). IV. CROSSING OVER AND NON-DISJUNCTION IN PLATYPOECILUS MACULATUS GÜNTH

A. W. BELLAMY
University of California, Los Angeles, California

Received February 7, 1933

In previous reports (BELLAMY 1928, 1933) the characters Rubra, Nigra, and Pulchra have been interpreted as gene mutations at a single locus on one of the heterosomes, although the possibility of close linkage was recognized (BELLAMY 1928, p. 229). Data presented by FRASER and GORDON (1929) indicate that Rubra, supposing that the Rubras in their laboratory and mine are of the same sort, very probably is a composite of the characters "Red" and "Spots." They observe the appearance of a red, non-spotted fish, which, because of its breeding behavior, was interpreted by them as due probably to crossing over between X and Y chromosomes. The separation of Red and Spots has not been observed in my laboratory but the appearance of certain other exceptional individuals favors the interpretation of crossing over between the X and Y chromosomes in this species.

Incidentally it is probable that it was the interpretation of the origin of this non-spotted red that led GORDON (1931, p. 764) so completely to misinterpret one of my statements (BELLAMY 1928) relative to the simultaneous occurrence of the Rubra and Pulchra patterns in females. Apparently GORDON assumes the Pulchra to be identical with Rubra minus the gene for red color. It is possible of course that Pulchra had its origin from the Rubra-type but I know of no direct evidence on that point. I do know, however, that the Rubra-Pulchra pattern has not been observed in my aquaria in females, and that a Rubra-Pulchra male (red and spotted) is fairly easily distinguished from Rubra (also red and spotted) in mature specimens. The occurrence of red, spotted (Rubra) females was described in my first report (BELLAMY 1924) and mentioned in so many places, in addition to detailed descriptions of their hereditary behavior, in the second report (BELLAMY 1928) that GORDON's statement that "BELLAMY (1928) has taken the curious position of doubting the existence of such a color combination (Rubra or red spotted platy) in the female," is just a little ludicrous.

The occurrence of non-spotted red as described by FRASER and GORDON raises the question of the genetic constitution of both Pulchra and Nigra in addition to Rubra. It is possible that they are composite patterns also but until they are resolved recognition is impossible.

If we suppose that Red and Spots had separate origins, Rubra arose, presumably from crossing over between two heterosomes. In this case Pulchra can be regarded as a Rubra less the gene *R*, FRASER and GORDON'S symbol *Sp* can be used for it, and a chromosome map represented as in figure 1. Stipples or White, is regarded as the wild-type and its symbol, +, is not included.

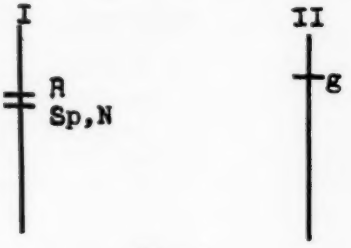


FIGURE 1

A cross of Pulchra male × Rubra female would then be written, following the Drosophila schema: $\frac{+Sp}{+Sp} \times \frac{RSp}{}$, and the fish described as a Rubra-Pulchra would be: $\frac{R\ Sp}{+Sp}$. The + symbol is used here in the same sense as FRASER and GORDON'S recessive *sp*.

A possible method for obtaining additional information, in the absence of additional crossing over, involves matings between Red, non-spotted and Pulchra (figure 2).

FIGURE 2.—Red, non-spotted $\frac{R+}{R+} \times \frac{+Sp}{}$ Non-red, spotted (Pulchra)

Red, spotted $\frac{R+}{+Sp}$ $\frac{R+}{}$ Red, non-spotted

The red, spotted males, $\frac{R+}{+Sp}$ should be substantially identical with typical Rubras and therefore distinguishable from the typical Rubra-Pulchra males obtained from crosses of Rubra males and Pulchra females. As previously noted Pulchra-Rubra males have a more extended pattern of macro-melanophores than typical Rubras. The spots tend to be larger and are more numerous.

The next question involves the relation of Nigra to Red, *R*, and Spots, *Sp*. *N* might be regarded as a pattern factor complementary to *Sp* or simply as allelomorphic to *R*.

If *N* is allelomorphic to *Sp*, and is a complete unit in itself, sooner or later crossing over will effect a relatively stable combination of *R* and *N*. Such a fish, if encountered in nature, in the absence of special information would be regarded as a single unit as was the case with Rubra before FRASER and GORDON observed the appearance of the non-spotted red fish. If *N* is complementary to *Sp*, crossing over would result in *N* and *R* genes entering one gamete, + and *Sp*, another, the latter fertilizing an egg containing the Y chromosome of the female, leaving the vacant (as regards *N* and *Sp*) X chromosome to be fertilized by an X bearing sperm carrying the wild-type gene.

If *N* is allelomorphic to *R* and crossing over occurs in the female between the X and Y chromosomes, a separation of *N* from *Sp* can result, and the event would be similar to the separation of *R* from *Sp* as assumed by FRASER and GORDON (1929, p. 171). The other crossover described by FRASER and GORDON (1929, pp. 168 *et seq.*) differs in that both *R* and *Sp* are supposed to have entered the Y chromosome together.

Crossing over of the type indicated above can explain the appearance of the exceptional Wild-type female recorded in mating 89, table 3 (BELLAMY 1928). Since the Nigra pattern results from a definite organization of macro-melanophores, presumably a fish having the gene for *N* but not for *Sp* would be a reversion to the wild-type. The appearance of this exceptional female is accounted for, however, equally well by assuming that both *N* and *Sp* entered the Y chromosome of the female, as indicated above. Likewise the three exceptional Wild-type females appearing in mating 44.2.1, table 3, (BELLAMY 1928) are accountable for in a similar manner. If this is crossing over, it has occurred four times among 962 offspring in matings such that detection was possible. This amounts to approximately 0.4 percent. Since, in this case, only half of the crossovers are detectable, the indicated value is approximately 0.8 percent, a value that need not be taken too seriously until more data are available.

The exceptional Wild-type individual recorded in mating 317 (tables 4, 6, BELLAMY 1933) requires a different explanation. This individual was born October 20, 1923, and on March 20, 1924, it was recorded as a white (Wild-type) heterozygous for Gold. On April 15, 1924, it was isolated as an immature male since it showed the typical thickening and pseudo-segmentation of the third anal ray. On May 21, 1924, it was noted on the records that the fish had made "no further progress toward maleness. Third anal ray shows only a thickened condition with segmentation extending into distal third." Since the male parent was a homozygous Rubra, crossing over in the female cannot account for the production of a Wild-type of either sex. Non-disjunction in the male, however, and fertilization of an egg by a sperm having no X chromosome would produce a zygote having

either an X or a Y chromosome, depending upon which type of egg was involved. The fact that the fish did not develop into a normal individual of either sex further indicates an abnormal distribution of sex factors.

SUMMARY

1. The bearing of FRASER and GORDON's (1929) findings is discussed in relation to the genetic constitution of Pulchra and Nigra. It appears probable that Pulchra is identical with FRASER and GORDON's "Spots."

2. A value somewhat under one percent is indicated for crossing over between X and Y chromosomes in the female.

3. An exceptional Wild-type inter-sex (?) is interpreted as resulting from non-disjunction in the male.

LITERATURE CITED

BELLAMY, A. W., 1928 Bionomic studies on certain teleosts (Poeciliinae). II. Color pattern inheritance and sex in *Platypoecilus maculatus* Günth. Genetics **13**: 226–232.

1933 Bionomic studies, et cetera. III. Hereditary behavior of the color character, Gold. Genetics **18**: 522–530.

FRASER, A. C. and GORDON, MYRON, 1929. The genetics of Platypoecilus. II. The linkage of two sex-linked characters. Genetics **14**: 160–179.

GORDON, MYRON, 1931 Morphology of the heritable color patterns in the Mexican killifish, Platypoecilus. Amer. J. Cancer **15**: 732–787.

AN EXPERIMENTAL AND THEORETICAL STUDY OF CHROMATID CROSSING OVER[1]

MARCUS M. RHOADES
Cornell University, Ithaca, New York

Received February 17, 1933

INTRODUCTION

The term "crossing over" is used to denote the exchange of pieces or segments between homologous chromosomes. There are many facts which indicate that the exchange of parts or segments occurs during the first meiotic prophase when the two homologous chromosomes are in intimate association. Prior to 1916 crossing over was thought to take place between the two paired chromosomes before they had divided equationally. BRIDGES, however, in that year, from his study of non-disjunction of the X chromosome of *Drosophila melanogaster* came to the conclusion that each chromosome was split equationally when crossing over occurred. Later L. V. MORGAN (1925), ANDERSON (1925) and BRIDGES and ANDERSON (1925) in a beautiful series of experiments substantiated BRIDGES' earlier conclusion; and ANDERSON (1925) and BRIDGES and ANDERSON (1925) further demonstrated that only two of the four strands crossed over at any one level. This was true for both diploids and triploids. REDFIELD (1930, 1932) working with triploid Drosophila found that the II and III chromosomes likewise crossed over when each chromosome was split equationally. ANDERSON used the phrase "four strand crossing over" to denote the divided condition of the chromosomes at the time crossing over took place in diploids. This term should not be applied to triploids, since it infers diploidy, even though only two of the three chromosomes (four chromatids) are involved in any one point of crossing over. The terms "double strand" or "chromatid crossing over" could be used for both diploids and polyploids since they refer only to the presence of the equational split.

In addition to the chromosomes of *D. melanogaster*, the X chromosomes of *D. simulans* (STURTEVANT, 1929) and *D. virilis* (DEMEREC, unpublished) have been genetically proved to cross over at a double strand stage. RHOADES (1932) presented data which proved that the *pr*-v_2 chromosome in Zea, the fifth largest in the monoploid complement, crossed over after, or at the time, the equational split occurred. WHITING and GILMORE (1932) reached a similar conclusion for one chromosome of Habrobracon in their study of impaternate daughters from virgin females. As far as the writer is aware, these are the only cases where chromatid or double strand crossing

[1] Paper No. 194 from the Department of Plant Breeding, Cornell University, Ithaca, New York.

over has been genetically demonstrated. (LINDEGREN recently [1933] demonstrated chromatid crossing over in the fungus Neurospora.) There are certain data (BLAKESLEE and others 1923, FROST 1931) in plants which can be interpreted as the result of double strand crossing over, but since other explanations are possible they can not be said to prove that double strand crossing over occurred.

In recent years many cytological papers by various investigators have dealt with the nature of the tetrad present in the first meiotic division of diploid organisms. They have attempted to discover the cytological mechanism by which genetic crossing over is effected through their study of the nature and origin of chiasmata. There is much controversy regarding many salient points, but most of the investigators agree that only two of the four chromatids in each chiasma are involved in the actual exchange of partners.

STERN (1931) and CREIGHTON and MCCLINTOCK (1931) have proved that genetic crossing over is accompanied by an actual exchange of parts between the chromosomes. Later (1932) CREIGHTON and MCCLINTOCK presented cytological evidence that crossing over in Zea takes place between chromatids. Zea thus becomes the first organism in which chromatid crossing over has been demonstrated both genetically and cytologically.

The occurrence of chromatid crossing over in such diverse forms as Drosophila, Habrobracon, and maize suggests that it may be a wide spread or universal phenomenon. However, WETTSTEIN'S data on Funaria, where he found only two types of spores among the quartets from sporophytes which were heterozygous for linked factors, indicates that crossing over in Funaria takes place between undivided chromosomes and not between chromatids.

DOUBLE STRAND CROSSING OVER IN ZEA

In diploid organisms where the four chromatids which comprise the tetrad are normally distributed to the quartet of cells arising from each meiocyte and where it is impossible to recover the four resulting cells from any given meiocyte it is impossible to tell genetically whether crossing over occurs in a single or double strand condition. It was only through such aberrant behavior as non-disjunction which results in two of the four chromatids going to a single member of the quartet that the occurrence of double strand crossing over was genetically proven in diploid Drosophila. In trisomic and polyploid individuals where the number of homologous chromosomes present in metaphase I makes it possible for some members of the quartet to regularly receive more than one chromosome it is possible to test for the occurrence of double strand crossing over.

For this study in Zea the *pr*-v_2 trisome, which involves the fifth largest

chromosome, was used since these trisomic plants differ markedly in appearance from their disomic sibs and an accurate classification into the two classes is possible.

The writer, in 1932, published a preliminary note on the genetical demonstration of double strand crossing over in Zea. This paper will present more extensive data upon this subject.

The factor pair *Pr:pr* differentiates between purple and red aleurone color and the factor pair V_2:v_2 is responsible for green and virescent seedling color. These two pairs of genes give a recombination value of 41 percent as shown by the data in table 1. The crossover value or map distance

TABLE 1

Control data for percent of recombinations between pr and v_2 in diploids. Pr V_2 pr v_2×pr v_2.

PEDIGREE	PURPLE ALEURONE GREEN SEEDLING	PURPLE ALEURONE VIRESCENT SEEDLING	RED ALEURONE GREEN SEEDLING	RED ALEURONE GREEN SEEDLING
1831–1840	438	267	288	375

40.6 percent of recombinations.

is much greater than this since undetected double crossovers reduce the percent of recombinations.

Trisomic plants of $\frac{Pr \quad V_2}{Pr \quad V_2}$ constitution were

$$\frac{Pr \quad V_2}{pr \quad v_2}$$

pollinated by double recessive individuals. The ensuing seeds were classified into purple and red aleurone classes and when planted the seedlings classified as green or virescent. Classification into trisomic and disomic types was made just before anthesis.

If crossing over among the members of the trivalent occurred between undivided chromosomes and not between chromatids there should be no cases of trisomic plants homozygous for *pr* or v_2. If, however, crossing over took place between chromatids the occurrence of trisomic plants homozygous for the two loci is expected. Diagram 1 shows how a trisomic plant homozygous for v_2 may arise. Similarly if a crossover had taken place between *pr* and the spindle insertion, a trisomic homozygous for *pr* would be possible. The data in table 2 show 61 trisomic individuals homozygous for the v_2 gene. There were nine plants among the 553 individuals arising from red aleurone (*pr*) seed which were trisomic. Only two of the 15 pedigrees listed in table 2 failed to throw exceptional trisomic types.

The constitution of the trisomic plants was *AACCRr* with respect to the aleurone factors affecting color. Since the male parents were *AA*

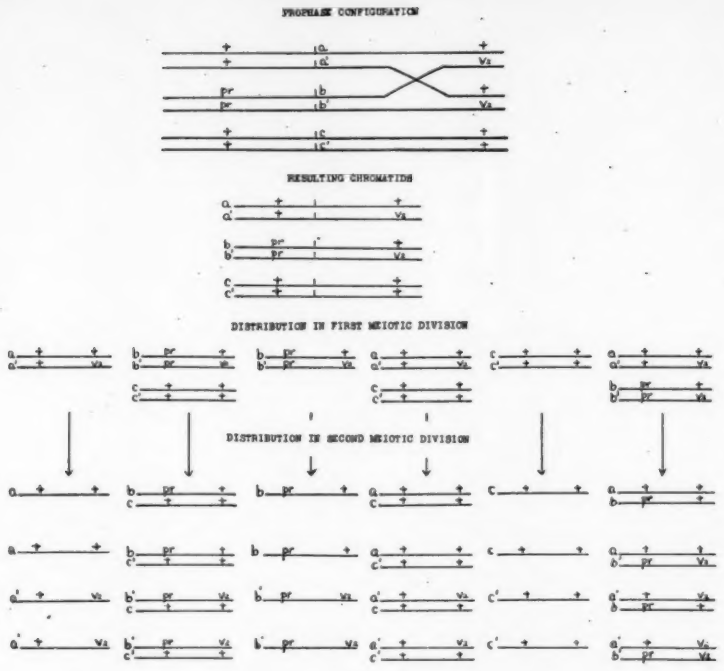


DIAGRAM 1

Diagram illustrating how a trisomic plant homozygous for v_2 may arise through chromatid crossing over. The combination in the lower right hand corner will give rise to the exceptional trisomic type.

CCRR it was expected that all the seeds would have colored aleurone and could be classified as purple or red. Surprisingly enough approximately 50 percent of the seeds had colorless aleurone. Subsequent investigation showed that two of the *r* genes were dominant over a single *R* gene. This *r* allelomorph has been designated r^{rw} (the superscript *r* to represent its effect upon plant color and the superscript *w* to represent its effect upon aleurone color). This behavior is analogous with the floury-flinty endosperm situation. Although the *Pr:pr* constitution of these colorless seeds could not be told without testing, they were planted since data on the v_2 locus could be had.

Since approximately only one-half of the seeds could be classified for aleurone color the total number of trisomic plants homozygous for *pr* should be twice 9, or eighteen. This is a legitimate procedure since the *R* locus is in another chromosome. Five of the nine *pr* trisomes were also

TABLE 2

Data on cross of $\frac{Pr \quad V_2}{Pr \quad V_2}$ *trisomes by double recessive male parents.*
$\frac{}{pr \quad v_2}$

PEDIGREE	CULTURE	ALEURONE	2N GREEN	2N v_2	2N+1 GREEN	2N+1 v_2
1482 (1)×1055	1620	235 *Pr*	79	23	72	4
	1621	279 wh.	95	47	73	3
	1622	59 *pr*	24	27	0	0
1482 (2)×1055	1623	20 *pr*	2	12	0	0
	1624	146 wh.	55	38	19	0
	1625	115 *Pr*	49	12	24	0
1482 (3)×1055	1626	157 *Pr*	41	15	65	2
	1627	43 *pr*	15	25	0	0
	1628	132 wh.	46	32	28	3
1482 (4)×1055	1629	40 *pr*	9	27	0	2
	1630	169 *Pr*	65	17	50	2
	1631	175 wh.	74	32	40	3
1482 (5)×1055	1632	231 *Pr*	93	21	68	5
	1633	237 wh.	78	50	53	2
	1634	59 *pr*	20	24	0	1
1482 (6)×1055	1635	41 *pr*	15	20	0	0
	1636	133 *Pr*	57	13	38	1
	1637	201 wh.	76	28	52	0
1482 (7)×1055	1638	186 *Pr*	65	15	70	3
	1639	51 *pr*	16	28	0	0
	1640	171 wh.	69	36	33	3
1482 (8)×1055	1641	185 *Pr*	65	24	43	1
	1642	65 *pr*	14	30	1	0
	1643	268 wh.	84	50	60	6
1482 (9)×1055	1644	36 *pr*	9	23	0	1
	1645	146 wh.	50	27	48	1
	1646	124 *Pr*	44	11	50	1
1482 (10)×1055	1647	194 *Pr*	87	19	57	3
	1648	56 *pr*	21	24	0	0
	1649	253 wh.	106	50	48	1
1482 (11)×1055	1650	60 *pr*	12	28	2	1
	1651	228 *Pr*	62	10	66	3
	1652	281 wh.	99	46	49	1
1582 (12)×1055	1653	34 *pr*	8	8	0	0
	1654	170 wh.	52	34	21	0
	1655	121 *Pr*	23	15	28	0
1482 (13)×1055	1656	34 *pr*	7	20	1	0
	1657	131 *Pr*	50	12	46	1
	1658	179 wh.	55	37	55	2
1482 (14)×1055	1659	227 *Pr*	82	27	71	1
	1660	266 wh.	116	47	50	4
	1661	64 *pr*	21	36	0	0
1482 (15)×1055	1662	34 *pr*	12	8	0	0
	1663	111 wh.	38	14	20	0
	1664	126 *Pr*	50	4	38	0

homozygous for v_2. These individuals might have arisen through non-disjunction at any one of the four divisions following the first meiotic division. But the normal frequency of non-disjunction during these divisions is so low that this interpretation can be disregarded. The v_2 trisomes from purple aleurone seed all proved to be $\frac{Pr \quad v_2}{pr \quad v_2}\frac{}{pr \quad v_2}$ in constitution. The male pronucleus brought in a single $\frac{pr \quad v_2}{}$ chromosome so the egg must have been $\frac{Pr \quad v_2}{pr \quad v_2}$ in constitution.[1] The origin of eggs of this type is possible only through chromatid or double strand crossing over. The same argument holds for those *pr* trisomes of $\frac{pr \quad V_2}{pr \quad v_2}\frac{}{pr \quad v_2}$ constitution. Here the egg must have been of $\frac{pr \quad V_2}{pr \quad v_2}$ constitution and this combination is possible only through chromatid crossing over. The genotypic constitutions of all the exceptional trisomic plants are presented in table 3. This table

TABLE 3

Genotypic constitutions of exceptional trisomes listed in table 2.

PLANT	GENOTYPIC CONSTITUTION	$2N+1$ BY ROOT TIP COUNTS	$2n+1$ BY ALEURONE OR SEEDLING RATIOS	$2n+1$ BY APPEARANCE
1620 (2)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1620 (3)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1620 (A)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1620 (B)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1621 (1)	v_2 v_2 v_2	"	—	"
1621 (2)	v_2 v_2 v_2	"	—	"
1621 (A)	v_2 v_2 v_2	"	—	"
1626 (A)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1626 (1)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1628 (1)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1628 (2)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1628 (3)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1629 (1)	*pr pr pr* v_2 v_2 v_2	"	—	"
1629 (2)	*pr pr pr* v_2 v_2 v_2	"	—	"
1630 (2)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1630 (3)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1631 (1)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1631 (2)	v_2 v_2 v_2	"	—	"
1631 (3)	v_2 v_2 v_2	—	—	"

[1] Extensive tests made by the writer show that pollen carrying an extra *pr*-v_2 chromosome rarely, if ever, functions in competition with haploid pollen since of a total of 1845 plants from the cross of a disome by a trisome there were no trisomic plants.

TABLE 3 (*Continued*)

PLANT	GENOTYPIC CONSTITUTION	$2N+1$ BY ROOT TIP COUNTS	$2n+1$ BY ALEURONE OR SEEDLING RATIOS	$2n+1$ BY APPEARANCE
1632 (1)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1632 (2)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1632 (5)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1632 (6)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1632 (7)	*Pr* ? *pr* v_2 v_2 v_2	—	—	"
1633 (1)	*pr pr pr* v_2 v_2 v_2	"	"	"
1633 (2)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1634 (1)	*pr pr pr* v_2 v_2 v_2	"	—	"
1636 (1)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1638 (2)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1638 (A)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1638 (B)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1640 (A)	*Pr pr pr* v_2 v_2 v_2	—	"	"
1640 (B)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1640 (C)	v_2 v_2 v_2	—	—	"
1641 (1)	*Pr* ? *pr* v_2 v_2 v_2	—	"	"
1642 (2)	*pr pr pr* V_2 v_2 v_2	—	"	"
1643 (3)	*Pr* ? *pr* v_2 v_2 v_2	"	—	"
1643 (2)	v_2 v_2 v_2	"	—	"
1643 (B)	*Pr* ? *pr* v_2 v_2 v_2	—	"	"
1643 (C)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1643 (D)	v_2 v_2 v_2	—	—	"
1643 (E)	v_2 v_2 v_2	—	—	"
1644 (1)	*pr pr pr* v_2 v_2 v_2	—	—	"
1645 (1)	*pr pr pr* v_2 v_2 v_2	"	"	"
1646 (1)	*Pr pr pr* v_2 v_2 v_2	—	"	"
1647 (1)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1647 (2)	*Pr pr pr* v_2 v_2 v_2	—	"	"
1647 (3)	*Pr* ? *pr* v_2 v_2 v_2	—	—	"
1649 (1)	v_2 v_2 v_2	—	—	"
1650 (1)	*pr pr pr* v_2 v_2 v_2	"	"	"
1650 (2)	*pr pr pr* V_2 v_2 v_2	"	"	"
1650 (3)	*pr pr pr* V_2 V_2 v_2	"	"	"
1651 (1)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1651 (2)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1651 (4)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1652 (1)	v_2 v_2 v_2	—	—	"
1656 (1)	*pr pr pr* V_2 v_2 v_2	"	"	"
1657 (2)	*Pr* ? *pr* v_2 v_2 v_2	—	—	"
1658 (A)	*Pr pr pr* v_2 v_2 v_2	—	"	"
1658 (B)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1659 (1)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1660 (A)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1660 (B)	*Pr pr pr* v_2 v_2 v_2	"	"	"
1660 (C)	v_2 v_2 v_2	—	—	"
1660 (D)	v_2 v_2 v_2	—	—	"
1660 (1)*	*pr pr pr* V_2 v_2 v_2	"	"	"

* From table 4.

TABLE 4

The genotypic constitution for the pr locus in non-virescent trisomes from colorless aleurone seeds.

PEDIGREE	NUMBER OF PLANTS OF *Pr Pr pr* CONSTITUTION	NUMBER OF PLANTS WITH *Pr pr pr* CONSTITUTION	NUMBER OF PLANTS WITH *pr pr pr* CONSTITUTION	TOTAL
1621 et cetera to 1663	62	106	1	169

shows that two of the v_2 trisomes from colorless seed were also homozygous for *pr*. One hundred and sixty-nine non-virescent trisomes from colorless seed were either selfed or backcrossed by *A C R pr* plants. In either case a 1:1 ratio for colored:colorless aleurone resulted. One of these 169 plants was homozygous for *pr*. Therefore it is apparent that the doubling of the number of *pr* trisomes was justified in order to find the approximate number of *pr* trisomes in the total population.

The genotypic constitutions of the exceptional trisomic types listed in table 3 were determined by crossing with the appropriate testers. For example, the constitutions of the v_2 trisomes with respect to the *pr* locus were determined by selfing and out-crossing with *A C R pr* individuals. Plant 1626–1 was a trisomic v_2 plant from purple seed. That it had the constitution $\frac{Pr \quad v_2}{pr \quad v_2}$ is shown by the following tests: (1) It was a trisome by root tip counts and appearance. (2) When self-pollinated it gave 177 *Pr*:95 *pr* seeds which is close to a 2:1 ratio. (3) When used as the male parent on *A C R pr* silks it gave 62 *Pr*:122 *pr*, a good 1:2 ratio. (4) It was homozygous for v_2 as shown by its appearance and when it was crossed with v_2 plants gave only v_2 individuals in the F_1. Another example is as follows: Plant 1630–2 was a trisomic v_2 plant from purple seed. When selfed it gave 39 *Pr*:17 *pr* seeds and when used as the male parent in a backcross gave 118 *Pr*:234 *pr* seeds. It was trisomic by appearance and root tip counts, and tests showed it to be v_2. These results are typical of those obtained for the other exceptional trisomes and they seem to make possible the statement that chromatid crossing over in Zea is a proven fact.

While the writer feels that a perfectly accurate classification into trisomes and disomes can be made by appearance alone, an attempt was made to secure root tip counts for all of the exceptional trisomic plants. These counts have been included in table 3.

Among the trisomic individuals listed in table 2 there were 4.1 percent of them homozygous for v_2. If the locus of v_2 is far enough removed from the insertion region so that its distribution is at random with respect to the insertion region the percentage of trisomic plants homozygous for v_2

should be 6.7. It would seem therefore that the locus of v_2 was some distance (crossover distance—not physical distance) from the spindle fiber. If 18 be accepted as the approximate number of *pr* trisomes, this gives a percentage of 1.2. The locus of *pr* then should be much closer to the insertion region than is the locus of v_2 since the frequency with which a gene becomes homozygous is a function of its crossover distance from the spindle fiber.

Five of the nine *pr* trisomes were also homozygous for v_2 while four were non-virescent. If *pr* and v_2 were closely linked and on the same side of the spindle fiber they would be expected to appear together in the exceptional trisomic types. But since the two loci are far apart (41 percent recombinations) there should be no tendency for the *pr* trisomes to be v_2, irrespective of whether or not they are in the same arm of the chromosome. One of the four green *pr* trisomes was of $\frac{pr \quad V_2}{pr \quad V_2}$ constitution. The egg was therefore $\frac{pr \quad V_2}{pr \quad v_2}$ in constitution. This suggests that *pr* and v_2 are on opposite sides of the insertion region since a much simpler type of prophase configuration will give the above combination if *pr* and v_2 are in different arms than if they are in the same arm. This is in agreement with the genetic map of this chromosome which places the gene bm_1 between *pr* and v_2 and McCLINTOCK (1932) believes bm_1 to be near the insertion. The cytogenetic data of the writer on a reciprocal translocation involving this chromosome also places *pr* much nearer the insertion than is v_2.[2]

THE ORIGIN OF A 32 CHROMOSOME PLANT

In the winter of 1931–32 a planting of seed of exactly the same cross as reported in this paper was made for the purpose of demonstrating the occurrence of chromatid crossing over. Since it was not considered possible to identify all of the trisomic plants in the seedling stage these data have not been included in table 2. They were, however, the basis for the statement by the writer in 1932 that crossing over occurred in Zea between chromatids. One of the v_2 plants from a purple seed in these cultures was classified as a trisome. Root tip counts in several clear figures showed that this plant had 32 chromosomes. If the pollen contributed 10 of these chromosomes there is left a total of 22 chromosomes as the contribution from the egg. The plant was clearly v_2 as progeny tests showed later,

[2] Data recently obtained from a study of this translocation give, in conjunction with McCLINTOCK's placing of the bm_1 locus, the following order: bm_1—insertion region—*pr*—v_2.

so it is possible to say that an n+1 megaspore, with two chromosomes carrying the recessive v_2 gene as a result of chromatid crossing over, in some way doubled its number of chromosomes to 22. Whether this occurred through non-disjunction of the entire chromosome set during the formation of the embryo sac or through fusion of the egg nucleus with a synergid it is impossible to state. The fact remains, however, that a doubling of the chromosome number occurred during the gametophytic generation.

TRISOMIC RATIOS

If a trisomic plant of *Pr Pr pr* constitution is pollinated by recessive pollen a 5:1 ratio of *Pr*:*pr* is expected if 50 percent of the eggs are n+1. Since only 31 percent of the progeny from a plant trisomic for the *pr*–v_2 chromosome are trisomes the theoretical ratio of *Pr*:*pr* is 3.35:1. If, however, we assume that crossing over occurs in a double strand stage and that the locus of the factor under observation is sufficiently far from the spindle fiber region so that it assorts at random with respect to the spindle fiber, the theoretical ratios are markedly changed. With 50 percent of trisomes and chromatid crossing over with randomness a 4:1 ratio of *Pr*:*pr* is expected instead of a 5:1 ratio with "chromosome" crossing over. Since only 31 percent of the plants are trisomes a ratio of approximately 3:1 (25.1 percent recessives) for *Pr*:*pr* is expected with chromatid crossing over and random assortment. It follows that the genetic ratio for a given factor pair in triploid individuals depends in part upon the location of the gene with respect to the point of spindle fiber attachment. That is, the further removed (crossover value) the locus of the genetic factor is, the greater the effect, up to the ratio expected with random assortment, will be upon the ratio of dominants to recessives. In other words if the locus of a factor is close to the insertion region its genetic ratio will be little affected by the occurrence of chromatid crossing over while a progressively greater disturbance in the genetic ratio will occur the further removed the gene lies from the insertion region. Thus the theoretical gametic ratio in duplex trisomic individuals with 31 percent of the progeny trisomes will be 3:1 with chromatid crossing over and random assortment while it will be 3.35:1 with "chromosome" crossing over. Therefore, the ratio of dominants to recessives in the progeny of triploid individuals should indicate whether the gene in question is close or far removed from the insertion region.

Since we have calculated the theoretical ratios expected on the various assumptions let us see what the observed ratios were for the two factor pairs reported in this paper. The percentage of *pr* seeds among a total progeny of 14,160 was 22.7 and the percentage of v_2 plants in a total of 4856 individuals was 24.9. The expected percentage of recessives from a

duplex trisomic individual with 31 percent of its progeny trisomes and "chromosome" crossing over, or with chromatid crossing over and the location of the gene near the insertion, should be 23 (3.35:1 ratio). This suggests that *pr* should be close to the insertion region. With 31 percent of trisomes and chromatid crossing over with random assortment the expected percentage of recessives should be 25.1. There were 24.9 percent of v_2 plants which indicates that the locus of v_2 is far enough removed from the insertion so that an approach to a random assortment is realized. The observed percentages of *pr* and v_2 agree very well with the position of these loci with respect to the insertion region as determined from the frequency of homozygous trisomic types.

The difference between 25.1 percent and 23.0 percent, the two extremes, is small and a large amount of data would be necessary to permit any definite conclusions as to the locus of a gene. If 50 percent of the functioning eggs were n+1 the difference in the percentage of recessives would be greater since 16.7 percent would be expected with chromosome crossing over as contrasted to 20 percent with chromatid crossing over and random assortment.

CROSSING OVER IN TRIPLOIDS

A theoretical discussion of the effect of double strand crossing over on crossover values in triploids and trisomics will be taken up in this section.

Represent the three homologous chromosomes present in a triploid or trisome as a, b, and c and let c carry the recessive genes. Crossing over can occur between a and b, a and c, and b and c with equal frequencies. Crossing over between a and b cannot be detected since both chromosomes carry the normal allelomorphs but crossing over between a and c, and b and c lead to detectable crossovers. Therefore, it can be argued that the amount of actual crossing over is 3/2 the observed amount (REDFIELD 1930). If there is a random distribution of the three chromosomes the proportion of crossover to non-crossover chromosomes should be the same in both the haploid and diploid eggs. REDFIELD (1930, 1932) apparently assumed this for she multiplied the observed crossover values determined from the diploid progeny for the factor 3/2. If we make a similar calculation for the *pr*-v_2 region from the data in table 2 we find that the observed recombination value among the disomic offspring is 26.2 percent. The corrected value would be 39.3 percent which agrees very well with the recombination value of 40.6 percent found in the diploid controls. Since only the diploid offspring were used in the calculation, a similar recombination value, if crossing over is the same in mega- and microsporocytes, should be found if the trisomic plants were used as the male parent in a backcross with double recessive individuals. Here all the offspring can be used since none

of the n+1 pollen succeeds in effecting fertilization. A corrected recombination value, using the factor 3/2, of 41.0 percent was found.

In the discussion above it has been assumed that crossing over between a and c, and b and c lead to detectable crossovers. Let us examine the consequences of crossing over between b and c. For simplicity, we will assume that we are dealing with a rod-shaped chromosome and that the location of the two genes under observation is near the terminal insertion region. Represent the genes as *x* and *y* and the normal allelomorphs by the conventional + sign. The constitution of the triploid before crossing over takes place is:

+	+ a
+	+ a′
+	+ b
+	+ b′
x	*y* c
x	*y* c′

Crossing over occurs between chromosomes b and c but involves chromatids b′ and c. The constitution following the crossing over is:

+	+ a
+	+ a′
+	+ b
x	+ b′
+	*y* c
x	*y* c′

If the distribution of the three chromosomes is at random in the metaphase of the first meiotic division (arbitrarily assumed to be reductional for the spindle region) there are six combinations possible at the end of the first division. They are as follows:

(1)		(2)		(3)		(4)		(5)		(6)	
+	+ a			+	+ a			+	+ b		
+	+ a′			+	+ a′			*x*	+ b′		
		+	*y* c			+	+ b			+	+ a
		x	*y* c′			*x*	+ b′			+	+ a′
+	+ b			+	*y* c			+	*y* c		
x	+ b′			*x*	*y* c′			*x*	*y* c′		

The next division is equational for the spindle region so 12 of the resulting 24 combinations have 2 chromosomes represented and the other 12 have a single chromosome. But 4 of the 12 single chromosome types are crossover chromosomes and 8 are non-crossovers and 8 of the 24 chromosomes in the "two chromosome" combinations are crossover strands. That is, instead of every crossover between b and c leading only to detectable cross-

over chromosomes we find that only one-third of the recovered chromosomes are crossovers whereas in diploids one-half of the recovered strands, from every crossover point, are crossovers. Therefore, in place of using the factor 3/2 to obtain the actual amount of crossing over it is necessary to use the factor 2.25.

If the factor 2.25 is the correct one to use and the factor 3/2 used by REDFIELD is wrong, then her comparisons between triploid and diploid crossing over must be revaluated since her triploid values were obtained by multiplying the observed crossover values by 3/2.

In a comparison of crossing over in triploids and diploids it is necessary to state the basis upon which the comparison is to be made. In diploids the frequency of crossover points in a short region is always twice the map distance since only one-half of the strands exchange segments at the crossover point. Therefore, instead of saying that genes A and B are 10 map units apart it would be equally proper to say that 20 cells out of every 100 have a crossover point between the loci A and B. We can then express map distances in terms of the number of crossover points as well as the percentage of crossover chromosomes among the progeny. In diploids a map distance of 10 means a crossover point frequency of 20 percent. If we wish to compare crossing over in a certain region in triploids with that in diploids we must keep in mind that what we really want to measure is the frequency with which crossover points occur in that region. We know that in triploids only two of the three chromosomes are synapsed at a given level, with the third chromosome acting as an univalent (cytological observations on plant sporocytes), while the Drosophila genetic data show that crossing over occurs between only two of the three chromosomes at any level. Therefore, in a short region we can have only one crossover point in triploids and only one in diploids. If we wish to compare the total amount of crossing over for a given region in triploids with that in diploids we must bear in mind that what we have to measure is the frequency with which crossover points actually occur in that region rather than the frequency with which crossover strands are found in the progeny. Since the proportion of crossover chromosomes recovered from a crossover point in triploids is not the same as in diploids, as shown on page 546, this difference must be taken into account. The writer believes that the proper way to "compare the actual amount of crossing over in triploids with that in diploids" is to compare the actual frequency of crossover points in the two forms rather than the resulting frequencies of types observed in the progeny. In other words, if we are to reach a real understanding of crossing over, we should study the mechanism involved and not confine our attention to the results of its action. It is upon these grounds that it is suggested that REDFIELD failed to use the proper correction factor, since

she explicitly stated that she was making the corrections to obtain the actual amount of crossing over in triploids.

All of the foregoing calculations have been based on the assumption that there was a random distribution of the three chromosomes in the first meiotic division. What would be the consequences if there should be a correlation, either positive or negative, between crossing over and disjunction? The data presented in this paper do not aid in solving this question but the Drosophila triploid data can be utilized. BRIDGES and ANDERSON (1925) studied crossing over in the X chromosome of triploid Drosophila. The three X chromosomes carried mutant genes so situated that the identity of a considerable portion of any of the recovered strands could be established. Approximately 41 percent of the progeny from a triploid mother are diploid females whose two X chromosomes come from their mother. These females are XXY in constitution and are called exceptional daughters since both of their X chromosomes came from their mother. These exceptional daughters were mated and the constitution of their X chromosomes determined from their male offspring. The analysis of the genotypic constitution of the exceptional daughters permitted a calculation of the amount of crossing over which occurred in the triploid mothers. These crossover values were then compared with the values found for the same regions in the diploid controls.

Comparison between triploid and diploid crossing over for the X chromosome (after BRIDGES *and* ANDERSON *1925)*

REGION	TRIPLOID CROSSOVER VALUE	DIPLOID CROSSOVER VALUE	RATIO $\frac{T}{D}$
1	14.3	6.9	2.07:1
2	11.3	22.8	0.50:1
3	3.9	10.1	0.39:1
4	8.2	16.2	0.51:1

For the rightmost regions (nearest the spindle fiber) the ratio of triploid to diploid crossing over was about 1:2, while for the leftmost region the ratio was approximately 2:1. These ratios point to a real difference in the amount of crossing over in triploid and diploid females. But it is important to remember that the triploid values were calculated from those eggs which had received two maternal X chromosomes.

REDFIELD (1930, 1932) studied crossing over in the II and III chromosomes in triploid females. The amount of crossing over in the triploids was based on the constitution of those eggs which received a single chromosome from the mother. The calculated amounts of crossing over, using the correction factor 3/2, in the triploid for the various regions studied are compared below with the crossover values for the diploid control females.

Comparison of triploid and diploid crossover values for the II chromosome (after REDFIELD *1932)*

REGION	TRIPLOID CROSSOVER VALUE	DIPLOID CROSSOVER VALUE	QUOTIENT $\frac{T}{D}$
al-dp	8.3	10.0	0.83
dp-b	16.2	27.2	0.59
b-pr	7.4	5.7	1.30
pr-c	27.1	19.2	1.41
c-px	13.0	22.1	0.59
px-sp	4.4	5.7	0.77

Comparison of triploid and diploid crossover values for the III chromosome (after REDFIELD *1930)*

REGION	TRIPLOID CROSSOVER VALUE	DIPLOID CROSSOVER VALUE	QUOTIENT $\frac{T}{D}$
ru-h	19.5	25.3	0.77
h-th	14.9	15.3	0.97
th-st	1.2	0.4	3.00
st-cu	21.2	5.6	3.79
cu-sr	14.6	14.0	1.04
sr-es	6.1	8.9	0.69
es-ca	18.0	34.3	0.52

The location of the spindle fiber in the II chromosome is slightly to the right of *pr* while the spindle fiber is situated between *st* and *cu* in the III chromosome. Therefore, we see that in REDFIELD'S experiments where her calculations of triploid crossing over are based on the constitution of those eggs which received a single chromosome the calculated amount of crossing over near the insertion region in triploids is higher than for the corresponding regions in diploids. The relative amount of crossing over in triploids in the distal regions of the II and III chromosomes is less than in the diploid controls. These results are the converse of those found by BRIDGES and ANDERSON. Since REDFIELD'S calculations were based on those eggs which received a single strand while BRIDGES and ANDERSON'S were obtained from those eggs which received two strands it seemed plausible to the writer that there might be a direct relationship between crossing over in triploids and the distribution of the members of the trivalent group. Especially did this seem likely since ANDERSON'S (1929) data showed that non-disjunction of the two X chromosomes in diploid females was more likely to happen if there was little or no crossing over between the two chromosomes. To tell if there is a correlation between crossing over and disjunction in triploids it is best to study both those eggs which receive a single strand and those which receive two strands. Unfortunately the published data do not permit such a direct comparison but the data of BRIDGES and ANDERSON on the types of association in the two chromosome combinations do permit some tentative conclusions to be drawn.

We will for the present confine our interest to the rightmost regions

(nearest the spindle fiber) studied by BRIDGES and ANDERSON. Assume that a crossover occurs between chromosomes a and b near the insertion region. We know that only one of the chromatids from chromosome a and only one chromatid from chromosome b are involved in the crossover. The two chromatids from chromosome c are not involved in any crossover in this region (BRIDGES and ANDERSON). The identity of the six strands following the crossing over is as follows: a a

b a′

a′ b

b′ b′

c c

c′ c′

If we assume that crossing over near the insertion region has no effect on the distribution of the chromosomes, then six types of combinations are possible at the end of the first meiotic division (we are arbitrarily assuming that reduction occurs at the first division since it is immaterial whether the first or second division is reductional for the insertion region). After the second (equational) division 24 combinations are expected with equal frequencies. The "two chromosome" combinations containing crossover strands can be classified into the following types of association:

Types of association		*Frequency*
crossover	b a	4
dissimilar non-crossover	c c	
crossover	b a	2
similar non-crossover	b b	
complementary crossover	b a	1
complementary crossover	a b	

The single chromosome combinations are composed of 4 crossover to 8 non-crossover strands and the percentage of crossover strands is 33.3 among the single strands. There are twelve "two chromosome" combinations which comprise a total of 24 strands. Eight of these are crossover chromosomes so the percentage of crossover strands is here also 33.3 Therefore if the distribution of the three chromosomes (six chromatids) is not influenced by crossing over near the insertion region the same amount of crossing over should be found in both types of eggs.

But assume that crossing over near the insertion region does have an effect on the distribution of the chromosomes and, since ANDERSON's 1929 data gives such an indication, further assume that if two chromosomes undergo crossing over near their insertion regions they always pass to

different poles. As before the identity of the six strands following the postulated crossover is:

a	a
b	a′
a′	b
b′	b′
c	c
c′	c′

But since the mode of disjunction has been determined by the previously occurring crossover we have only four instead of six combinations at the end of the first division. And instead of 24 combinations only 16 are expected at the end of the second division. The "two chromosome" combinations containing crossover strands can be resolved into the three types of association:

		Frequency
crossover	b a	4
dissimilar non-crossover	c c	
crossover	b a	0
similar non-crossover	b b	
complementary crossover	b a	0
complementary crossover	a b	

There are 16 strands in the "two chromosome" combinations and 4 of these are crossover chromosomes which is a percentage of 25.0. Among the single strand combinations the number of crossover strands is 4 out of a total of 8 or a percentage of 50.0. Obviously if there is a correlation between crossing over near the insertion and distribution, it makes a great difference in the observed crossover values which class of eggs are studied. With complete correlation and using those eggs which received two chromosomes the amount of crossing over would be only one-half that which would be found if the eggs with only one chromosome were used. BRIDGES and ANDERSON found the amount of crossing over in the rightmost region in the triploid mother was only one-half that in the diploid. Their calculations were based on those eggs which received two chromosomes. REDFIELD, working with eggs which received a single chromosome, found the amount of crossing over in the regions near the spindle fiber to be from one and one-half to more than three times as much as in the diploid controls. It would seem that such differences might have some relation to crossing over and the manner of disjunction.

In table 12 of the paper by BRIDGES and ANDERSON (1925) are listed the various associations of crossover chromosomes from triploid females. As stated before if there is no correlation between crossing over

and disjunction there should be for regions near the insertion region a ratio of $4\frac{b\ a}{c\ c}:2\frac{b\ a}{b\ b}:1\frac{b\ a}{a\ b}$ combinations. With complete correlation the ratio should be $4\frac{b\ a}{c\ c}:0\frac{b\ a}{b\ b}:0\frac{b\ a}{a\ b}$. The data of BRIDGES and ANDERSON for their rightmost region, the right end of which is approximately ten units from the fiber, show $23\frac{b\ a}{c\ c}:1\frac{b\ a}{b\ b}:3\frac{b\ a}{a\ b}$ combinations. This is far from the 4:2:1 ratio expected with random disjunction and strongly supports the idea that there is a positive correlation in triploids between crossing over near the fiber and disjunction to opposite poles. In fact the correlation may be complete for regions very near the fiber as the deviation from the 4:0:0 ratio for the rightmost region may well be due to the fact that it was some 10 units from the end. Their data also show that for the leftmost region the approximation to a $4\frac{b\ a}{c\ c}:2\frac{b\ a}{b\ b}$ ratio is very close, which suggests that the effect of crossing over on disjunction is dissipated progressively away from the fiber. This would be expected.

If there exists a positive correlation between crossing over and disjunction the association of strands in the diploid eggs would be such that the percentage of exceptional daughters homozygous for a recessive gene whose locus is near the fiber end would be low, approaching zero as a limit. But since the effect of crossing over on disjunction becomes less away from the spindle fiber attachment point the percentages of exceptions homozygous for genes in the distal end should increase progressively. Only 1.1 percent of the exceptional daughters in BRIDGES' and ANDERSON'S data were homozygous for point V (*f*, *B*, +), approximately 10 units from the fiber, while 11.5 percent of them were homozygous for point I (*y*, *sc*, +), which is about 70 units from the fiber attachment point. RHOADES (1931) in a study of homozygosis in diploid females with attached X's, where crossing over cannot affect disjunction since that is predetermined, found 19.0 percent of the exceptional daughters homozygous for yellow (*y*) and about 5 percent of homozygosis for forked (*f*). The ratio of homozygosis for *y* and *f* in the attached X data is 3.8:1 while it is 10.4:1 in the triploid data. It is possible that this difference is due, in part at least, to the correlation between crossing over and disjunction in triploids. The marked differences in coincidence values in triploids and diploids found by BRIDGES and ANDERSON should also affect the frequencies of homozygosis.

Although the relation of crossing over to disjunction is admittedly more complex in V shaped chromosomes than in rod shaped it seems to the writer that the regional differences in crossing over between triploids and

diploids reported by REDFIELD and by BRIDGES and ANDERSON cannot be accepted until the possible relationship between crossing over and disjunction has been considered, and if the writer is correct in his argument, until the proper factor for undetected crossovers has been used. The correction factor for the distal regions in REDFIELD'S data should then be 2.25 instead of 1.5. We must assume, however, that there is for these regions no effect of crossing over on disjunction. For those regions near the insertion and where a strong correlation between crossing over and disjunction presumably exists the correction factor is 1.5. The difference in the correction factors for the distal and proximal regions is due to the correlation between crossing over and disjunction which results in a higher percent of crossovers in the proximal regions going to the haploid eggs. The correction factors for intermediate regions lies somewhere between these two values.

The above correction factors are to be used when only one of the three chromosomes is marked by mutant genes. If all three of the chromosomes are properly populated with mutant genes so that crossing over can be detected between all of the three homologues, different correction factors must be used. The correction factor for regions near the insertion becomes 1.0 while for distal regions, where no correlation exists between crossing over and disjunction, the correction factor should be 1.5. These correction factors are to be used when those eggs which receive a single chromosome are studied. If, as BRIDGES and ANDERSON did, those eggs which receive two chromosomes are studied, and all three of the chromosomes are marked by mutant genes, the correction factor for regions near the insertion should be 2.0 and for the distal regions it should be 1.5. If these correction factors are applied to BRIDGES' and ANDERSON'S data the amount of crossing over near the insertion is approximately the same in triploids as in diploids, while the amount of crossing over in the leftmost region of the X chromosome becomes even greater in triploids than in diploids.

The corrected percent of recombination of 39.3 for the *pr*–v_2 region was determined from the diploid offspring listed in table 2 of this paper but the correction factor used in obtaining this value was 1.5. As pointed out in a preceding section it is in close agreement with the percent of recombination found in the disomic controls and would seem to permit the conclusion that the amount of recombination in this region was approximately the same in trisomes and disomes. But if the factor 1.5 is not the one to use, as the writer argues, the corrected value of 39.3 is incorrect and a recalculation must be made using the proper correction factor. Since v_2 is some distance from the insertion region the correction factor should probably be much nearer 2.25 than 1.5.

ADDENDUM

Recently MATHER (1933) attempted to calculate the frequency of chiasmata in triploid Drosophila. He used crossover values from REDFIELD'S data which were based upon the constitution of the diploid offspring. His calculations led him to conclude that there was an excess of crossover chromosomes among the diploid progeny. To account for this calculated excess of crossovers he postulates that in triploids the three homologous chromosomes are associated as a trivalent group in two-thirds of the cases and in the remaining one-third as a bivalent and an univalent. Since crossing over can only occur between the members of the bivalent, and the univalent will pass at random to either pole, this would lead to an excess of crossovers among the diploid progeny.

The writer agrees with MATHER that there is an excess of crossovers among the diploid progeny. He does not, however, entirely agree with MATHER'S explanation of their occurrence although it is both possible and probable that some of the excess crossovers are caused by the formation of some bivalents and univalents instead of trivalent groups. But before accepting MATHER'S explanation the following facts should be mentioned·

(1) The excess of crossovers among the diploid offspring could be at least partially accounted for by the correlation between crossing over near the insertion and disjunction.

(2) The data of BRIDGES and ANDERSON (1925) and REDFIELD (1932) show that the two types of double crossovers (recurrent and progressive) occur with approximately equal frequency. This suggests that the frequency with which the three homologous chromosomes have failed to synapse so as to form a trivalent group at metaphase I is low (assuming the occurrence of a univalent is due to its failure to pair with the other two homologues in the meiotic prophases).

(3) MATHER'S assumption of univalents being formed in $33\frac{1}{3}$ percent of the cases is not universally valid since the frequency of trivalents at metaphase I in maize plants trisomic for the $pr-v_2$ chromosome is approximately 90 percent.

SUMMARY

1. The occurrence of chromatid or double strand crossing over in Zea was genetically demonstrated by the determination of the genotypic constitution of certain trisomic types.

2. The frequencies of homozygosis for *pr* and v_2 indicate that v_2 is much further removed from the insertion region than is *pr*.

3. The effect of chromatid crossing over upon genetic ratios in triploids and trisomes is discussed.

4. Genetic proof was obtained for the occurrence of the doubling of the entire chromosome set in the gametophytic generation.

5. The genetic data obtained by BRIDGES and ANDERSON, and REDFIELD for triploid Drosophila is discussed with reference to a possible correlation between crossing over in triploids and disjunction. Certain data of BRIDGES and ANDERSON are presented in support of a positive correlation between crossing over and disjunction.

6. REDFIELD's treatment of her triploid data is discussed and the suggestion is made that she failed to use the proper correction factor for undetected crossing over. It is further suggested that the triploid data of BRIDGES and ANDERSON should also be corrected.

LITERATURE CITED

ANDERSON, E. G., 1925 Crossing over in a case of attached X chromosome in *Drosophila melanogaster*. Genetics **10:** 403–417.

1929 A case of high non-disjunction in Drosophila. Z. I. A. V. **51:** 397–441.

BLAKESLEE, A. F., BELLING, JOHN, AND FARNHAM, M. E., 1923 Inheritance in tetraploid Daturas. Bot. Gaz. **76:** 329–373.

BRIDGES, C. B., 1916 Non-disjunction as proof of the chromosome theory of heredity. Genetics **1:** 1–52, 107–163.

BRIDGES, C. B. AND ANDERSON, E. G., 1925 Crossing over in the X chromosomes of triploid females of *Drosophila melanogaster*. Genetics **10:** 418–441.

CREIGHTON, H. B. AND MCCLINTOCK, B., 1931 A correlation of cytological and genetical crossing over in *Zea mays*. Proc. Nat. Acad. Sci. **17:** 492–497.

1932 Cytological evidence for 4-strand crossing over in *Zea mays*. Proc. Sixth Int. Congress Genetics. **2:** 392.

FROST, HOWARD, 1931 Uncomplicated trisomic inheritance of purple versus red in *Matthiola incana*. Proc. Nat. Acad. Sci. **17:** 434–436.

MCCLINTOCK, BARBARA, 1932 A correlation of ring shaped chromosomes with variegation in *Zea mays*. Proc. Nat. Acad. Sci. **18:** 677–682.

MATHER, K., 1933 The relation between chiasmata and crossing over in diploid and triploid *Diosophila Melanogaster*. J Genet. **27:** 243-260.

MORGAN, L. V., 1925 Polyploidy in *Drosophila melanogaster* with two attached X chromosomes. Genetics **10:** 148–178.

REDFIELD, HELEN, 1930 Crossing over in the third chromosomes of triploids of *Drosophila melanogaster*. Genetics **15:** 205–252.

1932 A comparison of triploid and diploid crossing over for chromosome II of *Drosophila melanogaster*. Genetics **17:** 138–152.

RHOADES, M. M., 1931 The frequencies of homozygosis of factors in attached X females of *Drosophila melanogaster*. Genetics **16:** 375–385.

1932 The genetic demonstration of double strand crossing over in *Zea mays*. Proc. Nat Acad. Sci. **18:** 481–484.

STERN, CURT, 1931 Zytologisch-genetische Untersuchungen als Beweise für die Morgansche Theorie des Faktorenaustauschs. Biol. Zbl. **51:** 547–587.

STURTEVANT, A. H., 1929 The claret mutant type of *Drosophila simulans*: a study of chromosome elimination and of cell-lineage. Z. wiss. Zool. **135:** 323–356.

WETTSTEIN, F. VON, 1924 Morphologie und Physiologie des Formwechsels der Moose auf Genetischer Grundlage. Z. I. A. V. **33:** 1–236.

WHITING, P. W. AND GILMORE, K. A., 1932 Genetic analysis of synapsis and maturation in eggs of Habrobracon. Proc. Sixth Int. Congress Genetics **2:** 210–211.

INDEX

Page numbers printed in black-faced type refer to headings of articles or of sections. Names in lists of "Literature cited" are not included.

ABERLE, DE, S.B., 340, 380, 386
Acrydium arenosum, **159**, 160, 162, 164, 166, 169, 170
  Biology, **159**
  Color patterns, **159**, 166
    Bilateral line, 161
    Black pronotum, 161, 164
    Brown spot, 163, 164
    Chalky white pronotum, 161, 164
    Creamy white, 161
    Deep red brown, 163, 164
    Dominant, 160, **161**, 162, 164, 166, **169**, 170
    Dull yellow, 163, 164
    Genes for, **169**, 170
    Gray white stripe, 163, 164
    Inheritance, **159**, 160
    Light brown or orange striped femora, 163, 164
    Light gray pronotum, 163
    Light lemon yellow, 163
    Mahogany brown, 163
    Mottled gray or "wild-type," 161, 162, 164
    Reddish brown, 163
    Specked femora, 163, 164
    Tawny brown stripe, 163, 164
    Variable white spots, 163
    White flecked pronotum, 163
    White spot, 163, 164
    White spot femora, 163, 164
    White to brown fine line, 163
    Yellow stripe, 163
    Effects of exposure to continuous light on, **160**
    Factors, sex-linked, 169, 170
    Inheritance, 160
    Nymphs, **161**, 165
    Results of pairing factors for elementary dominant color patterns, **164**
ADKINSON, J., 129, 146
Agapanthus, 391
AGAR, W. C., 218
Agricultural and Mechanical College of Texas, **329**
Albinism, inheritance, in Holstein cattle, 473
Algae, 161
ALLEN, G. M., 481
ALLEN, W. J., 130
Allergy, **129**, 131–138, 140–146
  Asthma, 129–131, 134, 135, 138, 143, 144
  Bronchitis, 134, 135, 143, 144
  Cortical involvement, 131
  Dominant character, 130, 132, 138, 141–146
  Eczema, 129, 130, 133–135, 138–140, 143, 144
  Gall stones, 130
  Gout, 129, 130, 146
  Hay fever, 129, 130, 132–135, 138–140, 144
    Inheritance of, 133
  Headache
Allergy (Continued)
  Headache (Continued)
    Bilateral, 131
    Hemicrania, 131
    Paroxysmal, 131
  Identical twins, 144, 145
  Influence of environment, 133, 146
  Influence of heredity, 133
  Inheritance, **129**, 131, 134
  Migraine, **129**–136, 138–140, 143–146
    Inheritance, 145
    Sex-linked dominant, 130, **145**, **146**
  Symptoms, syndrome, 130
  Urticaria, 129, 130, 133–135, 138–140, 143, 144
Allium, 391
  triquetrum, 390, 393, 407
Aloe, 406, 409
  striata, 390
ALTENBURG, E., 20, 190
American Jersey Cattle Club, 416, 444, 451
Ancestry, 339, 356
ANDERSON, E. G., **53**, 58, 175, 261, 262, 278, 396, 402, 403, 407, 409, 411, 535, 548–555
ANDREWS, A. A., 386
Anemia, in man and animals, 371
Angiosperms, 99, 109
Animal Stock Breeding, Institute of, **117**
Anthocyanin, 332
  Distribution in cotton, 330, 331
  Effects of alkaline solution, 331
Antirrhinum, 518
Apoteltix eurycephalus, 159–161, 164–166, 169, 170
APPERLEY, F. L., 130
Armour's Livestock Bureau, 441
Arnold Arboretum, **53**, **121**
Asthma, bronchial, 129
Atavism in Jersey Cattle, 445
Aucuba, 309
AVERY, P., **487**, 506, 511

Bacteria, 30
BAILEY, P. G., 72
BALLS, W. L., 329-331
Balyeat Hay Fever and Asthma Clinic, **129**
BALYEAT, R. M., **129**–131, 135
BARFURTH, D., 75
BARLEY, **148**
  Allelomorphic series
    Defeciens, 148, 153
    Distichon, 148, 150, 153
    Vulgare, 149, 150
  Black Hulless, 149, 150, 154–157
  Chevalier, 149–153
  Colsess V, 148–156
  Dominant gene, 150, 153
  Fertility, 149
  Gene
    Awns, 148, 149, 151, 153, 154

Barley (Continued)
Gene (Continued)
Black glume, 149
Chlorina, 150, 158
Chlorina seedlings, 148, 149, 151, 157
Covered caryopsis, 151, 154
Dull green, 148
Green seedlings, 150, 151, 154–157
Inheritance, 150
Hoods, 148, 149, 151, 153, 154
Hulled, 148, 149
Intermedium, 149, 151, 153, 157
Long-haired rachilla, 148, 149, 151, 154–156
Naked, 148, 149, 154
Naked caryopsis, 151, 154
Non-intermedium, 149, 151, 153
Non-six-row, 148–150, 153, 156, 157
Normal green, 148, 149, 154
Purple straw color, 149, 150, 156, 157
Short-haired rachilla, 148, 149, 151, 154–156
Six-row, 148–150, 153, 156, 157
Two-row, 150, 153, 157
White, 148, 149
White glume, 149
White seedlings, 154–158
Inheritance, 150
White straw color, 149, 150, 156, 157
Yellow, 148, 149
Inheritance, 148–150
Intermedium factor, 150, 157
Mendelian factor pairs
Inheritance, 149
Interrelation, 150
Minnesota, 148
Nepal, 149, 151, 152
Nilsson-Ehle No. 2, 149, 150, 156
Straw color, inheritance of, 150, 158
Trebi, 148–150, 154–156
Virescent seedlings, 151
BARTH, L. G., 288
BARTLETT, H. H., 293, 310, 312
BATESON, W., 73, 75, 86, 91, 295, 389
BAUR, E., 129–131, 133, 324
BEADLE, G. W., 261, 263, 276, 278
Begonia, cytological examinations of, 324
BELAR, K., 121, 390, 391, 406
BELIAJEFF, N. K., 114
BELLAMY, A. W., 522, 523, 526, 531, 533
BELLING, J., 28, 53, 121, 308, 309, 312, 388–390, 405
BLACKBURN, K. B., 311
BLAKESLEE, A. F., 53, 60, 308, 309, 312, 518, 536
BLANK, E., 338
BOEDIJN, K., 293, 310, 312
BOLEN, H. R., 115
BOMAN, N., 450, 451
BOND, C. J., 211, 212
BORING, A. M., 68
BOVERI, 294
BOVIE, W. T., 47
Brassica napocampestris, 311
Breeding tests, 339, 342–344, 346–348, 357–359, 361–364
BREWSTER, W., 37
BRIDGES, C. B., 18, 19, 21, 22, 25, 43, 46, 47, 114, 115, 119, 173–176, 178, 179, 255, 256, 259, 261, 272, 273, 278, 284, 285, 535, 548–555
BRIEGER, E., 95, 106, 107, 109
BRINK, R. A., 53, 60
Brooklyn Botanic Garden, 78, 229
BUCHANAN, J. A., 129, 146
BURKART, A., 288
BURNHAM, C. R., 53, 60, 61, 63
Bussey Institution, 97, 324
Butyl alcohol, 98

Cadzow Castle Park, 457
California Institute of Technology, 173, 250, 284
California, University of, 221, 224, 230, 240, 388, 487, 493, 522, 531
Camera lucida, 199
Campanula, 53, 61–67, 308
Attempts at crossing, 324
Canary, 213
Canna, 308, 309
CAPINPIN, J. M., 308
Carnegie Institute of Washington, 278, 284, 388
Carnegie Year Book for 1930, 284
CAROTHERS, E. E., 121–123, 127
Carotin, 329, 332
CARVER, W. A., 330
CASEMENT, D., 478, 479
CASTLE, W. E., 221, 233, 243, 244, 335
CATCHESIDE, D. G., 64, 65, 308
Cattle, 238, 243–247, 415, 447, 471, 479
Aberdeen, 443, 445, 446, 466–468, 471
Albinism, inheritance, in Holsteins, 473
Angus, 223, 224, 229–237, 239, 240, 246, 443, 445–447, 451, 459, 466–469, 471–474, 477
Assortive mating, 429, 430, 432, 434, 435, 437, 439
Atavism, 445
Ayrshire, 224, 229, 230–237, 239, 240, 246, 443–445, 447–451, 457, 463, 469, 470, 473–475, 477
Genetic variability in, 224, 232
Halo, 475
Beef breeds, 222–226, 231, 232, 235, 237, 239–243, 245, 247, 248
Body conformation, 222–226, 238–241, 245–248
Body length, 416, 418, 420–423, 426–432, 435–439
Brown Swiss, 443, 478
Butter fat, 421
Color gene, 442, 473
Dilution factor, 447, 455, 456, 475
Dominant white, 457
Hereford pattern, 451
Intensity factor, 447, 469, 475
Minus modifiers, 451
Recessive white, 455
Sex-limited, 443–445
Color inheritance, 441, 442, 446, 450, 479
Conformation
Inheritance, 239
Relation to milk yield, 417
Correlation

Cattle (Continued)
Correlation (Continued)
Correlation between genetic constitutions of mates, 434, 436
Correlation between genotypes of different mates, 434, 436
Dam and daughter, 433, 437
Determination of heredity by dominance deviations, 433, 436
Determination of size by common environment, 433, 436
Determination of size by heredity, 433, 436
Fraternal, 416, 433, 439
Parent and offspring, 416, 433, 439
Sire and daughter, 433
Sire and his mates, 432
Dairy breeds, 222–225, 229, 231, 232, 235, 237, 239, 240, 242, 243, 246–248
Dairy type, 415, 420, 429, 435
Depth of withers, 417, 418, 420, 422, 423, 426–432, 435–438
Dutch Belted, 463–465
English Park, 457, 458
Environment, 224
Evolution of, 415, 439
Extension factors, 452
Fat, 232, 235, 237
Feeding, 224
Fleckviehs, 451, 458–460
Fleshing, 223
Friesians, 443, 445, 466, 473, 478
Galloway, 452–454, 456
Gene
Black, 443–458, 463, 468, 469, 471–479
Black-spotting, 443, 444, 463, 468, 470, 472
In newly born calf, 443
Black tongue in Jerseys, 444
Brindle, 444, 445, 446, 455–457, 469, 472, 475, 476
Inheritance, 446
Blue-gray, 452–455
Brockle-face, 462, 463, 472
Claret, 448
Cream color, 474
Dominant white-spotting
Colorsided, 465–467
Dutch belt, 463–465
Hereford pattern, 458, 464
Inguinal white, 467, 468, 471
Dun, 447, 448, 474, 477
Fawn, 455–457, 468, 469
Hornless, 145
Lineback, 462, 476
Undesirability for breeding, 462
Pied, 464, 465
Pigmented ear, 457
Pigmented leg, 450, 457, 462, 463, 472, 473, 477
Pigmented nose, 469, 470
Black, 470
Brown, 470
Pigmented skin, 443, 456, 469, 471
Red, 442–448, 451–456, 458, 459, 463, 469, 471–473, 476–479
Cattle (Continued)
Gene (Continued)
Red-eye, 461
Red-necked, 461, 462
Roan, 451–453, 454, 455, 476, 479
Silver-gray, 455, 456
White, 449–454, 455–461, 463, 465–467, 469, 472, 475, 476, 479
White-face, 463, 472
White-head, 451
White-spotting, 443, 444, 448–451, 453–455, 457–460, 464–468, 470, 471, 473–477
White switch, 451
Whitening, 468, 477
Yellow (dilute red), 447, 448, 476
General conformation
Mature cows, 238
Muscle-skeleton index in different breeds of all ages, 239
Genetic composition of various breeds, 471
Aberdeen-Angus, 471
Ayrshire, 475
Guernsey, 477
Hereford, 475
Holstein-Friesian, 473
Jersey, 474
Shorthorn, 476
Genetic variability, 439
Groningen, 462, 463, 465
Groniger Zwartblaard, 464
Growth factors
General, 244, 245, 247
Group, 244–247
Specific, 244–247
Guernsey, 224, 229–237, 239, 240, 246, 443, 445–447, 449, 450, 461, 463, 469, 470, 477, 478
Halo, 477
Heart girth, 416–418, 420, 422, 423, 426–432, 435–438
Height at withers, 222–226, 229–231, 233–235, 238, 241–248, 416–418, 420, 422, 423, 425–432, 435–438
Height in calves, 235, 236, 237
Height in mature animals, 229, 231, 233–235, 247
Hereford, 224, 229–237, 239, 240, 246, 445, 447, 448, 455–463, 468, 470, 472, 475, 476, 478, 479
Holstein, 224, 229–237, 239–241, 245, 246, 248, 443, 445, 447–451, 459, 460, 465–470, 473, 475, 477, 478
Inbreeding, 437, 439
Influence of dam's conformation on that of her daughters, 421
Influence of selective breeding, 416
Inheritance, 415, 416, 420, 435, 439, 441, 442
Effect of unlike factors in, 421
Jersey, 223, 224, 229–237, 239–243, 246, 247, 415, 416, 419, 424, 429, 431–433, 435, 437, 439, 443–447, 451, 455, 459, 468, 469, 472, 474, 475, 477
Ancestry and history of, 415
Genetic constitution of, 415, 439
Influence of environment, 415–417, 421, 425, 427, 433

Cattle (Continued)
Jersey (Continued)
Genetic (Continued)
Influence of inheritance, **415**, 417, 420, 425–428, 433, 437, 438
"Majesty" line, 469
Relative influence of heredity and environment on conformation, **433**
Lactation, 232
Lakenvelders (See Dutch Belted)
Limburger, 451
Maxwalton Sultan, 454
Mendelian factors, 421
Milk yield, 421, 429
Morphological similarity, 229
Muscle development, 224–226, 231, **233**, 234, **235**, 237, 248
Muscle diameter (See round measurement)
Muscle-skeletal index, 225, 239–242
Native Philippine, 455, 456
Nellores of India, 455–457
Norman breed of France, 463
Nymphomaniac, 230
Paunch girth, 416–418, 420, 422, 423, 426–432, 435–438
Pembroke, 457
Post-natal development, 242
Pregnancy, 232
Pre-natal development, **242**, 243, 247, 248
Red Danish, 445, 446
Relation between dam and her daughter, **422**
Relation between types of half sisters with common sire, **427**, **428**, **438**
Relation of conformation of daughter and dam, **423**, 424
Relation of conformation of full sisters, **425**, **426**
Relation of conformation of half sisters with common dam, **428**, **429**, **438**
Relation of conformation of sire and daughter, **417**, **418**–420
Environment, 419
Heredity for size, 419
Relation of height, muscle development and weight in mature animals of different breeds, **233**
Relation of size to age in assortive mating, 429
Round measurement, 223, **226**–228, **230**, 231–235, 237, 238, 241, 243, 245–247
In calves, **235**, **236**, 237
Rump length, 416, 418, 420–423, 426–432, 435–439
Schlesischen Landvieh, 478
Segregation, 222, 231
Selective breeding, 430, 431
Shorthorn, 224, 229–237, 239, 240, 242, 244, 246, 445, 447, 449–456, 459, 461, 462, 469, 470, 472, **476**, 478
Color inheritance, age effect on, 476
Size factors, **221**, 222, 238, 244–246, 248
Size inheritance, 221, 223, 239, 246, 425, 435, 437
Skeletal development (See height at withers)
Somatic color mutations, **478**, 479
South Wales, 457
Cattle (Continued)
Swedish breed of Stjernsund, **444**, 450
Swedish mountain breed, 457, 466
Swiss Simmental (See Fleckvieh)
Telemark (Norway) 446, 447, 466
Troenders, 466
Volume (See weight)
Weight, 223–225, 229, 230, **232**, **235**, 238, 416, 418, 420, 422, 423, 426, 428–432, 435–438
In calves, **235**, **236**, 237
In mature animals, 229, **232**, **233**–235, 238, 244, 245
West Highland, **447**, 457
Whitehall Sultan, 454
Width at hips, 416, 418, 420–423, 426–432, 435–439
Cattle Club, American Jersey, 416, 444, 451
CAVE, H. W., 441
Central Genetics Station of Narcomzem, 117, 118
Chicago, University of, 522
CHINO, M., **111**, 114, 115
Chlorophyll, 332
Chlorophyll deficiency, **148**
CHRISTIE, W., 92
CHRISTOFF, N., 96
Chromatid crossing over, **535**, 543–545, 554
Experimental and theoretical study of, **535**
Chromatin, functional, 5
CLARK, F. H., 338
CLAUSEN, R. E., 97, 311, 490, 498, 500, 502–504, 509, 515
CLELAND, R. E., 53, 60, 297
CLENCY, R., 159, 162
COLE, L. J., 446, 448, 468, 469, 472
COLLINS, G. N., 155
Colorado Agricultural Experiment Station, **148**
Congress, Sixth International Genetics, 193, 293
Conifers, polyploidy, 128
Constant hybrids, fertility, 311
Coolidge tube, 284
COOPER, D. C., 53, 60
Cornell University, **535**
Cotton, **329**, 330, 333
Anthocyanin distribution
Red leaf spot, 330, 331
Red plant color, 330, 331
Chlorophyll deficiency, **329**, 330
Egyptian, 329
Gene
Bronze, 331–333
Green, 329, **330**, **331**–333
Red, **329**–331, 333
Virescent yellow, **329**, **330**, **331**, **332**, 333
Yellow seedlings, 329
Green leaf, **331**
Inheritance, **329**
Inheritance of plant color, **329**
Mebane, 330
Red leaf, **330**, **331**, **332**
Self-fertilization, 332
Upland, 329
Virescent yellow, history and description, **330**
CRANE, M. B., 193

CREIGHTON, H. B., 388, 536
Crepis, 307, 309
CREW, F. A. E., 70, 452
CROWDER, 131
CROWTHER, J. A., 1, 5
Crustacea, crossing over in Gammarus, 93
CUTLER, D. W., 214
Cypripedium pubescens, 391
Cytology, **251**, 252

DAANE, A., 148–150, 154
Dahlia, 309
DALAND, G. A., 47
DANFORTH, C. H., 338
DARK, S. O. S., 407
DARLINGTON, C. D., 59, 61, 62, 64, 65, 121, 122, 308, 309, 313, 407, 515
DARWIN, 294
Datura, 53, 60, 61, 66, 207, 307–309, 389, 394, 410
  Nubbin, 518
  Reversed crossing over, 388, 404
  Stramonium, 312
DAVENPORT, C. B., 70, 75, 211, 212
DAVIS, B. M., **293**, 294, 298, 300, 306, 310, 313
DEAN, M., 159
Deletion, 179, 180
DEMEREC, M., 112, 116, 535
DEMING, G. W., 148, 149
Dendrocoelum, 389
DERMEN, H., 309
DESJARDINS, A. V., 40
DETLEFSEN, J. A., 473
DIEHL, D. G., 58
Differential viability, 402
Dimorphism, sexual, 70
Dispermy in animal material, 295
Dissection, 215
DOBROVOLSKAIA-ZAVADSKAIA, N., 338
DOBZHANSKY, SIVERTZEV-, N. P., **173**
DOBZHANSKY, T., 26, 46, 53, 63, **173**, 179, 180, 186, 188, 190, 251, 252, 261, 262, 278, 288, 389, 408
Dominance, incomplete, 129
DRINKWATER, H., 129
Drosera, 308
Drosophila, 5, 9, 15, 63, 115, 133, 134, 178, 180, 186–188, 190, 218, **250**, 261, 265, 269, 273, 275, 277, **284**, 532
  Abdominal tergites, disarrangement, 174, 183
  Aberrant, 32, 33, 35–37, 39, 40, 42, 45, 47–51
  Absence of crossing over in male, 93
  Blond translocation, 288
  Bobbed deficiency, cytology of, **178**
  Character, sex-linked, bobbed, 173
  Chromatin elimination, 46, 50
  Chromosome splits in sperm, 409
  Crossovers and chiasmas, 400, 401, 405, 407–409
  Experiments in crossing over, **112**
  Fertility, 3
  Frequency of chiasmata in triploids, 554
  funebris, 289
  Gene, **1**, 25
    Abnormal abdomen, 133
    Apricot, 33–41, 45, 49, 50, 250
    Ascutex, 21
Drosophila (Continued)
  Gene (Continued)
    Bar, 2, 23, 38, 190, 250
    Bar-eye, facets of, 134
    Beadex, 2, 24
    Beadex-2, 258
    Bent, 112, 114, 115, 119
    Black body, 134
    Bobbed, 33, **173**, 174, 178, 179, 181–183, 185, **188**–191, 252, **254**, 255, 258, **261**, 275
      Deficiency, **173**, **176**, 177, **178**–180, 182, 183, 185–189, 191
      Duplication, **173**, 177, **180**, 181, **182**–186, **188**–190
    Bristles, 174, 183–185
    Broad, 33, 181, 250, 253, 258, 287
    Bubble-wing, 287
    Carnation, 176, 253
    Chlorotic, 21
    Confluent wing veins, 286
    Crossveinless, 34–37, 41
    Curly-wing, 286
    Delta venation, 286
    Dichaete, 118
    Diminished, 46
    Dominant eyeless, 114
    Echinus, 21
    Echinus-like eye, 287
    Eosin, 284, 286, 287
    Extreme-bobbed, 174, 176, 177, 182, 183, 189
    Eyeless (Russian mutant allelomorph), 114, 115, 119, 120
    Facet, 21, 22, 36, 41, 44
    Forked, 21, 36, 41, 43, 250, 255, 258, 552
    Forked-3, 34–37, 41
    Furrowed, 21
    Fused, 21
    Gap, 111–113, 115, 116
    Garnet-2, 284, 286, 287
    Glossy, 111–116
    Gray, 39–41, 43, 49, 50
    Jagged-wing, 254
    Kurz, 181
    Lethal-VI a, 111, 116
    Lobe-2, 118
    Miniature, 2, 21, 24, 34–37, 41
    Miniature wing, 286
    Minute, 112, 285, 288, 289
    Minute IV, 114
    Mottled, 517, 519, 520
    Mottled-eye, 44
    Net, 112
    Non-bar, 185
    Non-bobbed, 181, 182, 253–255
    Non-spotted, 44
    Non-yellow, 185
    Notch, 286, 519
    Notched-wing, 44
    Pale, 112
    Parallel-sided wing, 173
    Pink-eye, 134
    Plain, 111, 116
    Plexate wing, 286
    Pointed, 284
    Prune, 181, 253, 258

Drosophila (Continued)
Gene (Continued)
Prune-like eye color, 287
Purple, 46
Red-eye, 523
Rotated abdomen, 114, 115
Rough eye, 173
Ruby, 21
Rudimentary-like wing, 287
Scute, 2, 33, 181, 250, 253, 255, 256
Scute-8, 33–41, 45, 49, 50
Shaven, 114
Short vein, 287, 289
Short wing, 173
Silver, 181
Singed, 21, 45, 286
Small bristles, 285
Small-wing, 2
Small-wing-2, 173, 175, **176**
Spotted, 44–46
Spread wing, 286
Star, 118
Star eye, 286
Stubby, 111–116
Stubby-2, 111, 116
Tan, 2
Truncate wing, 173
Vermilion, 2
White, 2, 34–37, 41, 44, 175, 253
White-eye, 45, 177
Wild, 114
Wild-fed, 134
Wild-type, 1, 27, 32, 33, 37, 523
Yellow, 21, 24, 33–41, 43–45, 48–50, 181, 182, 185, 250–256, 258, 261, 275, 287, 552
Gene rearrangement
Deletion, 520
Inversion, 520
Translocation, 520
Genes
Distribution of, in chromosome, **18–20**
Vital, 5, 6, 30
Germ plasm, 188
Gynandromorph, 32, 35, 36, 38, **41–43**, 47, 48, 51
Late hatching, 174
Lethals, 3–5, 8, 10, 17, 23–25, 27–29
Linkage tests, 44
melanogaster, 32, 114–117, 120, **173, 250,** 273, 278, 288, 290
Attachment of ends of closed chromosome, **252**
Bobbed deficiency
Cytology of, **178**
Interaction with bobbed duplications, **182**
Origin, **173**
Probable nature, **177**
Caucasus Belendzhik, 118
Chromosome IV, 115, **117,** 119
Crossing over in, 119, 120
Chromosome abnormalities, 288, 289
Comparison of closed and normal X chromosome, **258**
Cubitus, 117, 118, 120
Cubitus incompletus, 117

Drosophila (Continued)
melanogaster (Continued)
Cubitus interruptus, **117**
Genetic analysis, **118**
Double crossover frequency, **254**
Duplications for bobbed, **180**
Extra legs, 134
Fertility, 177, 254
Fertilization, 264, 267
Frequencies of all classes, **271**
Frequency of meiosis without exchange, **273**
Genovariation, **117,** 118, 120
Hereditary tumor, 133
Inversion, 177, 178
Inverted X, 269
Inviable crossovers, **261**
Inviable eggs, **269**
Linkage, 119
Mosaics due to fractional mutations, 286
Non-disjunction, 535
Normal Florida line, 117–119
Numbers of crossovers, 399, 411
"Oregon" stock, 173
Patroclinous males, **263**
Progenies of X-rayed males, 287, 290
Pupal stage, 255
Sister strand crossing over, **274**
Sterility, 250
Sterility in male, 289
Translocations, 177, 178, 180, 189, 190
Wild-type, **173,** 174, 176–186, **188–191,** 250
X chromosome, inert region of, **186–188,** 191
X-ray treatment, 289, 290
Mosaic, 34, 38, 39, 41–44, 45–48, 50
Eversporting, **44,** 45
Lethal, 48
Mosaic formation, **32,** 46
Mosaic types
Eversporting, 44
Females, **43**
Gynandromorphs, 41
Males, **45**
Mosaics
Experiments on treated females, **34**
Experiments on treated males, **38**
Mutant, 32, 34
Mutation
Dominant, 32
Dominant lethal, 3, 10, 14, 26–29
Dominant visible, 25, 27
Gene, 1, 4, 19, 21–24
Lethal, 10, 14–21, 23, 25, 26, 35–37, 39, 41 43
Recessive, 10, 32, 45
Viable, 20, 29
Visible, 19, 21–23, 27, 28, 30
Position effect, 190
pseudo-obscura, **284–288**
Abnormalities, **285,** 286, 289
Absence of testes, 285
Castrated males, 285
Deficiencies, **287, 288**
Fertility, **286**
Inert region, **287, 288**

Drosophila (Continued)
pseudo-obscura (Continued)
Mosaics, 286
Sterility, 286, 287
Venation plexus on one wing, 286
Wild-type, 284, 286
X chromosome length, 284
X-ray treatment, **284**, 286–290
Sex chromosome composition, 2, 18, 21–23, 26
Sex-mosaic, 32, 35–**41**, 47, 48, 51
simulans, crossing over in, 535
Sperm, 1, 4, 8, 11–13, 25, 27
Sperm head, 6–8
Spermatogenesis of, 187
Sterility, 3, 40, 45
Theta, 33, 43, 44, 47, 48
Translocations, 286, 287, 289
Reciprocal, 53
Viability of genes, 33, 34, 288
Viability region, 519
virilis, **111**, 113, **114**–116
Crossing over, **111**–116, 535
Dot-like chromosome, **111**, 115, 116
Experiments in crossing over, **112**
Lethal, balanced, 114
Mutants, **111**, 114, 115
Rod-shaped chromosome, 111
Sterility, 114
X chromosome genetical maps, 115
DUNN, L. C., 70, 72, 75, 77, 78, 82, 83, 88–92, 211, 221, 244, 448, 449, 481, 484
Dwarfness, 222

EAST, E. M., 96, 97, 108, 110, 324
ECKLES, C. H., 222, 226, 229, 237, 238, 247
Edwards Plateau, 55
EFROIMSON, W. P., 287
ELDERTON, 453
ELY, F. A., 130
EMERSON, S. H., 53, 263, 276, 278, 294, 297
Endocrine, sex, 129
Environment, 133, 134, 146
ERLANSON, E. W., 309
ESGUERRA, J. P., 455
EVVARD, J. M., 452–454
Experimental Biology, Institute of, **117**–119

Family history, 131, 132
Fertility, 3, 56, 57, 66, 108, 110, 149, 177, 327
Fertility of constant hybrids, 311
Fertilization, 46, 49, 95, 96, 104, 105, 107, 109
FETSCHER, R., 130
FISCHER, 129–131
FISHER, R. A., 433
Fixative
Allen's modification of Bouin's fluid, 97, 296
Allen-Bouin killing fluid, 199
Bouin solution, 7, 296
Brazilin, 493
Carnoy's solution, 493
Chromic-acetic-formalin, 390
Darlington's modification of Flemming's fixatives, 123
Flemming's mixture, 408
Iron-aceto-carmine, 493
La Cours', 123
Fixative (Continued)
Navaschin's fluid, 123, 179, 392, 408, 493
Strong Flemming, 296
Zenker's fluid, 296
Flies, 267, 269–271, 276
Flowering plants, **388**
Adjacent chiasmas, **397**
Attached and Ring X chromosomes
Attached X's and crossing over, **400**
Ring X's and crossing over, **403**
Break and join of chromonemas, **394**
Chromioles formed by secondary split, **393**
Crossing over in, **388**, **391**, 394, 407, 410
Direct chiasmas, **396**
Double chiasmas, 411
Gene rearrangement in, **388**, 410
Deficiency in, 389, **405**, 407, 410, 411
Deletion, 388, 394, **405**, 407, 410, 411
Insertion, 389, **405**, 407, 411
Inversion, 388, 394, **405**, 407, 410, 411
Reciprocal translocation, 388, **404**, 410
X-ray, caused by, 388
Reversed crossing over, 388, **404**, 410, 411
Translocation, 388, 394, **404**, 407, 411
Hypotheses, **389**
Hypotheses of crossing over
Gene rearrangement, **388**
Interchange, 394, 407, 410
Karyologic evidence, **390**
Oblique chiasmas, **397**
Opening-out at diplotene, **393**
Origin of chiasmas, **394**, 406, 410, 411
Division of chromomeres, **394**
Overlaps of homologues, **395**, 411
Proportion of chiasmas to crossovers, **398**–403
Smear preparations, 390, 391
X-ray as cause of point mutation, 409
FOLGER, 248
Food hypersensitiveness, 131
Fowl, **68**, 76, 77, 80, 83, 86, 88, 90, 92, 211, 212, 222
Ancona, 85
Autosomal factors, **68**, 71
Autosomal linkage, 77, 80, **82**, 83, 89
Backcross in female
Coupling phase, **85**
Repulsion phase, **83**
Backcross in male
Coupling phase, **85**
Black Langshan, 212
Black Minorca, 212
Black Orpington, 212
Brown Leghorn, 88, 212, 213
Buff Orpington, 212
Characters, association of, **76**
Chromosomal complex, **68**
Cochin, 72
Color (melanin), 83–86
Columbian, 212
Columbian Plymouth Rock, 210, **213**, **214**, 219
Crossing over, influence of sex on, **92**, 93
Dark Brahma, 212
Eye color, range and distribution of, 211
Gene
Bar, 210, 213, 218, 219

Fowl (Continued)
Gene (Continued)
Black, 71, 91, 212, 214
Black eye, 211
Black plumage, 211, 212
Blue, 78, 82, 91
Bluish-white shank, 213, 214
Brown eye, 212, 213, 215
Brown-in-down, 218, 219
Cerebral hernia, 78, 82, 83, 88, 92, 93
Comb, 212
Creeper, 68, 77, 78, 82, 89, 91
Crest, 69, **75**, 77–80, 82, 83, 87–90, 93
Dark blue shank, 210, **211**–213, 215, 219
Dark eye color, 210, 213, 215, 218
Dark iris color, 210, **211**–213, 219
Dominant white, 69, **76**, 78–80, 82, 83, 85, 88, 90, 93
Down color, 210, 212–214
Dark, 214, 216
Pale, 214, 216
Down striping, 214, 216
Ear-Lobe, 210, 212, 216
Egg color, 210
Fast feathering, 210, **211**, 213, 215–217, 219
Flecked, 212, 214
Frizzled, 82–87, 89, 93
Gold, 210, **211**–216, 219
Head-spot, 218, 219
Hen feathering in the male, 210
Leg-feathering, 69, **72**, 73, 77, 79, 80, 83, 91, 92
Light eye color, 210, 215, 218
Light iris color, 210, **211**, 212, 214, 216, 219
Light shank color, 210, **211**, 214–216, 219
Marked, 212
Muff and beard, 91
Multiple point comb, 92
Naked-neck, **69**, 70, 78–80, 82, 83, 91
Viability in, 70
No crest, 83, 88, 92
No inhibitor (of melanin), 83
Normal skull, 83
Pattern, 210, 214
Peacomb, 69, **74**, 79, 80, 83, 91
Plumage color, 212
Polydactyly, 69, **75**, 77–80, 82, 83, 90–92
Recessive white, 83, 91
Red, 84
Rose, 74
Rose comb, 68. 69, **73**, 77–80, 82, 83, **89**, 91–93
Rumplessness, 69, **70**, 71, 79, 80, 83 91
Shank color, 212–215, 218, 219
Silky, 91, 92
Silver, 210, **211**–217, 219
Single comb, 73, 74, 83, 89
Single-spike comb, 92
Skin color, **71**, 77, 212
Slow feathering, 210, **211**, 213–217, 219
Spangling, 216
Split comb, 78, 91
Tine-atine, 214
Walnut comb, 74
White, 212, 213, 456
Fowl (Continued)
Gene (Continued)
White shank, 83, **89**–91, 93
White skin, 69, 71, 72, 79, 80, 83–86
Yellow eye, 212–215
Yellow shank, 83, 89
Yellow skin, 71, 72
Gold Campines, 210, 212–**214**, 219
Hamburgh, 212
Inheritance in, 211, 212
Iris color
Duplex, 211
Simplex, 211
Triplex, 212
Langshan, 72
Light Brahma, 72–74
Linkage, 68, 76, **77**, **78**, **82**, 86, 87, 90, 93
Linked gene arrangement, **87**
Malay, 212
Pheasant, 214
Rhode Island Red, 84, **213**
Sebright, 212
Segregation, 212–**214**, 215
Sex chromosome, 68, 93, 216, **217**–219
Localization of, 210, **217**–219
Topography, **217**
Sex-linked genes, 82, **210**, 212–**216**, 219
Crosses made, **213**
Linkage relations, **215**, **216**
Silky, 212, 213
Silky Bantam, 72, 75
Silver Campine, 213, 214
Somatic segregation, 211
Spangled Hamburgh, 212, 213
White Leghorn, 76, 83, 221, 238, 244
White Orpington, 83–85
White Silkies, 456
Fragaria, 108
bracteata, 311
Helleri, 311
FRANDSEN, H. N., 311
FRASER, A. C., 522, 525, 526, 531–534
Fritillaria, 392, 408, 409
Sequence of primary and secondary splits, 389
FROST, H., 536
Funaria, 536
FUNKQUIST, H., 444, 450, 451

Gagea, 294
GAIRDNER, A. E., 59, 61, 62, 64, 65, 308, 324
Gallus domesticus (See Fowl)
Galton and Mendel Memorial Fund, 53, 121, 159, 250
Galtonia, 391
Gammarus chevreuxi, 115
GÄNSSLEN, 129, 131, 146
GATES, R. R., 293–295, 307, 308, 310–312
GAY, E. H., 1
GEERTS, J. M., 294, 308
GEIGY, R., 285
GELEI, J., 389, 390, 405
Gene number
Drosophila melanogaster, 410
Lilium, 410
Phrynotettix, 410
Genetical map, 115

Genetics, **82**, 88
Genetics, Central, Station of Narcomzem, 117, 118
Germination, methods of, **295**
GILMORE, K. A., 535
GONZALEZ, B. M., 455
GOODALE, H. D., 210
GOODSPEED, T. H., 97, 311, **487**, 488, 490, 502, 504, 506, 511, 513
GORDON, M., 522, 523, 525, 526, 531–534
GOSS, H., 243
Gossypium arboreum, 330
barbadense, 329, 330
hirsutum, 329–331
GOWEN, J. W., **1**, 269, **415**, 467, 468, 477
Grasshopper, 393, 410
GRAUBARD, M. A., 261
GRAVES, R. R., 223
GREEN, C. V., 222, 244–246, **481**
GREGORY, P. W., **221**, 243
GRIFFEE, F., 149
Grouse locust (See *Acrydium arenosum*)
GUILBERT, 248
Guinea-pig, 222, 484
Tortoiseshell, 473
GUYÉNOT, E., 269

HAAGE, 324
Habrobracon, 535, 536
HADLEY, P. B., 86, 91
HAIGH, L. D., 226, 232, 237
HAKANSSON, A., 308, 313
HALDANE, J. B. S., 210, 218
HAMMETT, F. S., 243
HANCE, R. T., 47, 68, 210
HANCOCK, 159
HANSON, F. B., 10, 15, 288
HARLAN, H. V., 149, 150
HARLAND, S. C., 329
HARRIS, 453
HARRIS, B. B., 288
HARRISON, J. W. H., 311
HART, 248
HARTWIG, N., **159**
Harvard University, 53, **95**, **324**, 335, 338
HAYES, H. K., 149, 150
HEITZ, E., 326
Hemerocallis, 308
Hemoglobin content, human standard, 379, 387
HERIBERT-NILSSON, N., 106, 293, 312
HERTWIG, P., 68, 210, 215–218
HEYS, F., 10, 15
Hibernation, 160
HOGE, M., 134
HOOPER, J. J., 444, 451
Hordeum distichon nigrinudum, 153
HORLACHER, W. R., **329**, 478, 479
HUETTNER, A. F., 46, 269
Human constitution, 435
HUMPHREY, L. M., **193**
HUNGER, F. W. T., 293
HUNT, H. R., **335**, 340, **367**, 370, 383
HUTT, F. B., **82**, 83, 91, 92
HUXLEY, J. S., 115
Hyacinthus, 307–309, 391
Hybridization, 95
Hydrocephaly in mouse, 338
Hypericum, 53
Hypothesis of modifying factors, 354

IBSEN, H. L., **441**, 450
ICHIJIMA, K., 311
IMMER, F. R., 151
Immersion oil, 392
Immunological relations, 106
INARIYAMA, S., 309
INGERSOLL, E. H., 159
Inheritance, **129**, **148**, 150, 157, **159**, **415**, **441**
Institute
Rockefeller, for Medical Research, **415**
Zoological, of Moscow State University, **117**
Institute of
Animal Stock Breeding, **117**
California, Technology, **173**, **250**, **284**
Experimental Biology, **117–119**
International Livestock Exposition of 1931, 472
Interrelation, **150**, 151, 154, 156, 157
Iowa State College, **193**
ISHIKAWA, M., 294

Jackson, Roscoe B., Memorial Laboratory, **481**
JANSSENS, F. A., 121, 122, 389
Janssens' hypothesis, 121, 122, 127, 128, 389, 405, 410
"Partial chiasmatypy," 121, 122, 127
Janssens' theory of crossing over
Objections and replies, **405**
Jersey, Cattle Club, American, 416, 444, 451
John Innes Horticultural Institution, 295, 298
Johns Hopkins University, 415
JONES (See MARSDEN-JONES)
JONES, S. V., 472
JORDAN, A., 130
JORGENSEN, C. A., 193, 199, 201–203, 207, 308, 309, 311
JULL, M. A., 72, 75, 77, 78, 82, 88–92

Kansas Agricultural Experiment Station, **68**, **159**, **441**, 448, 449
Kansas State College, 441, 472
KARPER, R. E., 330
KAUFMANN, B. P., 390
KAYE, G. W. C., 2
KEELER, C. E., 481
Kenilworth ivy (See *Linaria Cymbalaria*)
Kerchoff, William G., Laboratories of the Biological Sciences, 278
KIKKAWA, H., **111**, 114
KILLOUGH, D. T., **329**
KLEIN, A., 47
KNOX, C. W., 72
KOLTZOFF, H. K., 117
KOMAI, T., 116
KOONCE, D., 148, 149
KOOS, K., 193, 199, 309, 311
KOSTOFF, D., 95–97, 106–108
KRAFKA, J., 134
KRUMHOLZ, G., 95, 98
KUHLMAN, A. H., 232, 444, 474
KUIPER, K., 457, 462–465
KULENKAMPFF, H., 6
KULKARNI, C. G., 294, 296–298, 300, 306, 313

LAGSDIN, J. B., 1
LAMBERT, W. V., 72
LANCEFIELD, D. E., 284, 287
LANDAUER, W., 77, 82, 83, 88, 89, 91, 92
LANG, A., 451, 458
Larix, **121, 123,** 125, 127, 128
  Chiasma formation, **121**–123, 125–128
  Chiasma frequency in, 128
  Chiasmata and chromatids, **123**
  Crossing over, 121, 128
  decidua, 123, 125
  eurolepis, 123–125
  Kaempferi, 123–126
  Terminalization, 128
LARSON, I., **159**
Lathyrus odoratus
  Chiasma formation, 123
  Crossing over, 123
LAUPRECHT, E., 449, 473, 478
LAWRENCE, W. J. C., 309
LEAKE, H. M., 330
LEE, A., 435
LEFEURE, G., 213
LENZ, 129–131
LESLEY, J. W., 201, 203, 309
LESLEY, M. M., 199, 201, 203, 308, 309
LEVIT, S. G., 33
Levy-Hauser counting chamber
  Double, 373
  Single, 373
LI, J. C., 178, 188
Lilium, 391, 396, 408, 409
  Chiasma, **396, 397**
  pardalinum, 394, 395
  regale, 390, 392
  Sequence of primary and secondary splits, 389
Linaria, **324,** 327, 328
  aparinoides, 325, 326
  Artificial parthenogenesis, 327
  bipartita, 324, 327
  Broussonnetii, 325–328
  canadensis, 324
  Cymbalaria, 324–327
    alba, 324, 327
    compacta, 324, 327
    globosa, 324, 327
    Sexual incompatibility, 327
    vulgare, 324, 327
  dalmatica, 325
  delphinioides, 326, 327
  Fertility, 327
  Gene
    Carminea, 327
    Purple, 327
    Yellow-flowers, 328
  Gene for intensity, 327
  Genetic differences affecting flower color, 327
  Genetic observations, **324**
  genistifolia, 325
  Hendersonii, 325, 328
  heterophylla, 325, 326
  macedonica, 325
    nymphe, 325
    speciosa, 325
  macroura, 325–327
  maroccana, 325–327
Linaria (Continued)
  multipunctata, 325, 327, 328
  Pancicii, 326, 327
  Perezii, 325–328
  reticulata, 325–327
  sapphirina, 326, 327
  Sexual compatibility, 328
  Sterility of hybrids, 328
  Tournefortii, 326
  triornithophora, 326
  triphylla, 326, 327
  vulgaris, 325–328
LINDEGREN, 536
LINDSTROM, E. W., **193**–195, 199, 309, 311, 452
LINK, 325
Linkage trials, 92
LITTLE, C. C., 481, 482
Locust (Paratettix), crossing over, 93
LUSH, J. L., 445
LUTZ, A. M., 294, 307
Lycopersicum, 207
  esculentum, 193, **195**–197, 199–201, **202**–206, 208
    Character
      Flowers, 205
      Fruit, 205
      Leaf, 205
      Seed, 205
      Stature, 205
      Stem, 205
      Trichome, 205
    Chromosome doubling, 194
  pimpinellifolium, 193–**195**, 197–200, 202–206, 208
    Fruit size, 195, 197
    Variety
      Red Currant, 193, 195, 197
Lycoris, 309

MACARTHUR, C. G., 210
MACARTHUR, J. W., **210**
MCCANDLISH, A. C., 465
MCCLINTOCK, B., 53, 60, 179, 308, 309, 388–390, 393, 405, 407, 408, 410, 493, 536, 543
MCCLUNG, C. E., 121, 122
McClung theory, 121, 122, 127, 128
MCCRAY, F. A., **95**
MACDONALD, A., 248
MACDOUGAL, D. T., 293
MACDOWELL, 244
MCLENDON, C. A., 330
MAEDA, T., 121, 123
MAHONEY, C. H., 329
Maize, 218
  Crossing over, 536, 554
  Deficiencies, 393
  Genetics, 88
Mammal, 211, 213, 221, 222, **243**, 481
  Eye and hair color, interrelation of, 213
  Nature of growth factors, **243**
  Size inheritance, 222
Man
  Evolution, 415
  Forearm length, 433
  Span, 433
  Stature, 433
MANRESA, M., **455, 456**

MARSDEN-JONES, E. M., 311
MARSHALL, 325
MARTENS, P., 391
MASON, 112
MATHER, 554
MATHEWS, H. W., 448
Maturation, 46
MEAD, 224, 248
MENDEL, G., 390
Mendelian dominant, 523
Mendelian factor
  Inheritance in barley, 149
Mendelian factor pairs
  Interrelation, **150**, 151, 154, 156, 157
Mendelian genes
  Attraction in tetraploids, 207
Mendelian ratio, 495
Mendelian recessive, 500, 519, 526
Mendelizing difference, 21
Mercury vapor lamp, 160, 161
METZ, C. W., 112, 115, 187
MEURMAN, O., 309
Michigan State College, **335**, 338, **367**, 371
Michigan, University of, 58, **293**
MILLER, F. W., 223
Minnesota Agricultural Experiment Station, 82
Minnesota, University of, **82**
Missouri Agricultural Experiment Station, 232, 237
MIXTER, R., **335**, 340, 342–344, 357–359, 361, **367**, 370–373, 382–384, 386
MOFFETT, A. A., 309
MOHR, O., 46, 178, 179, 186, 487
Monobromide of napthalin objective, 410
MORGAN, L. V., 43, 179, **250**, 251, 261, 403, 411, 519, 535
MORGAN, T. H., 18, 19, 21, 22, 25, 43, 47, 114, 115, 133, 173, 174, 176, 178, 179, 285, 388, 399
Morphology, 27
Morus, 307
Mosaicism, 487
Moscow State University, **117**
MOSES, 112
MOULTON, C. R., 226, 232, 237
Mouse, 222, **335**, 342, 364, **367**, 372, 373, 379–381, **383**, 387, **481**
  Ancestry, effect of flexed, 356, **357**
  Anemia
    Hereditary, 340, 386
    In newborn, 340, 346, 349, 351, 367, 369, 380, 384
    Inheritance of, **367**, 368, **369**
    Recovery from, before birth, 385
  Backcross, **344**
  Breeding tests, 339, 342–344, 346–348, 357–359, **361–364**, 368
  Crossing over, 93
  Deficiency, 179
  Differential death rate, 353
  Distribution of offspring, **482**
  Environmental factors in embryonic development, 350
  Eye defects, 338
  Flexed×Flexed matings, **340**
  Flexed gene linkage relations, **362**
  Flexed×normal matings, **344**
Mouse (Continued)
  Flexed tail in, **335**, **337**, 339, **340**–343, **344**, **345**–348, **349**–359, 361–365, 367–**378**, 379–382, 383–387
    Deficiency in F_2 generation, 339, 340
    Inheritance, 338, 356, 359, 361, 368, 370
    Inheritance, grade of flexure, **355**
    Mendelian recessive, 338–340, 350, 351, 367, 368, 387
    Result of hereditary and environmental influences, 362
    Variability, 335, 338, 343, 348, 355, 358, 365, 380, 383
  Flexed tailed
    Anemia in, 338, 340–342, 344–346, 349, 350, 352, **367**, 368, 370, 371, 373, 377, 379–381, 383, 386, 387
    Blood of, **371**, 372, 374–382, 386
    Embryological development and morphology, 338
    Frequency distribution of hemoglobin, **378**, 386
    Frequency distribution of red blood cells at birth, **376**
  Gene
    Albino, 362–365
    Anemia, 383
    Bent-tailed, 338
    Black, 245, 482
    Brown, 245, 481, 482
    Chinchilla, 364
    Colored, 363, 364
    Dark-eye, 364
    Extreme dilution, 364
    Filiform-tailed, 338
    Flexed, 356, **362**, 363, 365, 371, 383
    Full color, 364
    Helicoid, 338
    Kinky-tailed, 338
    Non-shaker, 364
    Pink-eye, 364
    Shaker, 364
    Tailless, 338
    White, 340, 380, 386, 482, 485
  Genetic constitution of straight-tailed progeny of flexed crosses, **349**
  Genetic origin of flexed and anemic, **383**
  Growth factors, 244–246, 248
  Hereditary weakness, 348
  Heredity, 484, 486
  Inbreeding, 484
    Experiments, 340, 357–**360**, 361, 386
    Factor in suppressing reproduction, 358
  Inherited abnormalities, 355
  Mendelian mechanism of inheritance, 351
  Mortality of flexed, **386**
  Pale color in newborn flexed, 373, 379, 386
  Postnatal death rate, 347, 348, 365, 367
  Prenatal death rate, 347, 348, 365, 370, 371
  Short-tail, 340–344, 355, 357–360
    Inheritance of, 343, 355, 365
  Sterility, 358
  Uncertainty of classification of tails at birth, 384
  Uterine environment of normal controls, 377
  Ventral spotting, **481**–485
    Birth rank, influence of, 485, 486

Mouse (Continued)
Ventral spotting (Continued)
Influence of parents on, 483–486
Inheritance of, **481**, 484–486
Time of year, influence of, **485**, 486
Waltzing mouse, 338
White spotting
All white, 481
Black-eyed white, 481
Blaze, 481
Piebald, 481, 484
White on tail, 481, 482
White tail tip, 481, 482
X-ray experiments on germ plasm, 338
MULLER, H. J., 1, 15, 18–20, 22, 23, 27, 34, 114, 115, 179, 180, 186, 190, 261, 269, 284, 287–289, 388, 516, 520
MULSOW, 409
MURRAY, J. M., **481**
Mus bactrianus
Size inheritance, 244, 245
Mus musculus, **335**, **367**
Size inheritance, 244, 245

NABOURS, R. K., **159**, 160, 161, 164, 169, 170
NAGEO, S., 309
Narcissus, 309
Narcomzem, Central Genetics Station of, 117, 118
National Research Council, 487
NAVASHIN, M., 309
NAVILLE, A., 269
NEMEC, B., 294
Neurospora, 536
NEWTON, W. C. F., 121, 296, 309, 311
Nicotiana, **95**, 96, 99, 100, 106–109
brasilia, 96, 99–101, 103
Cuba, 107
digluta, 311
Embryo development, **95**
Fertility, 108, 110
Fertilization, 95, 96, 98, 104, 105, 107, 109
glauca, 96–103, 108, 109
Hybrids, **95**–97, 100, 102
Langsdorffii, 107
longiflora, 96, 100, 101, 109
nudicaulis, 96, 100–105, 109
Palmeri, 96, 100–102, 109
paniculata, 97, 99, 100, 102–105, 107–109
Pollination, 95, 97, 99, 100, 103
purpurea, 96, 100, 487, 489
Rusbyi, 96, 100–103, 107, 109
rustica, 96, 98–103, 105, 109
sanguinea, 97, 100, 102
Sterility, 108
suaveolens, 96, 99–105, 109
tabacum, 96–98, 100–105, 107, 109, **487**, 488, 493, 509
Attachment, 506
Fertilization, 506
Gene
Carmine, 499–501, 506, 513, 516, 519
Control, 489, 490, 495, 498, 499, 501, 503–506, 512, 513, 520
Coral, 499–501, 506, 513, 516, 517, 519
Deformed, **487**, 488, 490, 493–496, 498–506, 509, 510, 512–520

Nicotiana (Continued)
tabacum (Continued)
Gene (Continued)
Deformed (Continued)
Classification of progenies from, **491**, 496
Cytogenetics of, **487**
Cytological evidence of, **506**, 517
Derivation of progenies from, 490
Dissected limb, 488, 489
Flower lengths in populations, 497
Genetics of, **493**, 517
Progenies of normal from, 505
Tissue abnormalities, 490
Transmission of, 494
Transmission of *fluted* from, 501
Transmission of types derived from, 494
White-flecked, 488, 489, 506
X-ray derivative, **487**
Deformed and *carmine-coral* crossed with *coral*, 500
Enlarged, 504, 505
Fluted, 487, 490, 496, 498–506, 512–518
Large-lax, 498, 503, 505
Long-flowers, 490, 495, 496, 498–505, 512–516, 518, 519
Transmission of, 503
Mammoth, 490, 494–496, 502–506, 514–518
Normal, 490, 498, 500–503, 505, 506, 513, 515
Super-enlarged, 504
Super-long-flowers, 499, 504, 505, 515
Inheritance in, **487**
Restitution nuclei, 513
Sex-cell treatment by radium, 487
Sex-cell treatment by X-ray, 487, 488, 490, 506, 519
Variations, 488, 506
NILSSON-EHLE, H., frontispiece, xi 1

OEHMCKE, R. R., 448
Oenothera, **293**–295, 298, 303, 307–310, 312, 313
ammophila, 308
atrovirens, 293
Berteriana, 312
biennis, 293, 307, 308, 310, 311
biennis cruciata, 293, 307, 311
biennis cruciata gigas, 311
biennis cruciata gigas sulfurea, 293
biennis gigas, 311
Dwarfs, 312
Chromosome segregation, irregularities, 300, 308, 314
Cytology of triploid, **302**
Dispermy, origin of triploids through, 294, 295
Embryo size, 95, 98
franciscana, 293–298, 300–302, 304–310, 313, 314
Bud cone color
Green, 296
Red, 296
Cytology of tetraploid, **293**, 294, **305**, 308
Genetics of tetraploid, **293**, 294, 308
Inheritance

Oenothera (Continued)
franciscana (Continued)
Inheritance (Continued)
Color of bud cones, 296
Petal color, 296
Stature, 296
Tetraploid from, 293
franciscana gigas, 297, 299
Pollen size, 300
franciscana sulfurea nana, 295–298, 301, 313, 314
Gene
Broad bud cones, 300
Capsules short and thick, 300
Color
Sulfur, 296–298, 301, 313
Yellow, 296–299, 301, 313
Large flowers, 300
Leaves large and thick, 300
Narrow leaves, 299, 301, 304
Small flowers, 299, 301, 304
Stature
Dwarf, 296–301, 304, 312, 313
Tall, 296–298, 301, 313
Genetics of triploid and tetraploid, 296
Germination, 295, 298, 314
gigantea (Swedish), 312
gigas, 293, 307, 308
Fertilization, 294
gigas nanella, 293
grandiflora, 293, 310, 312
Hero plants, 294, 297, 298, 310, 313
hirtella, 293
Hypothesis of chromosome doubling, 312
Independent segregation, 297
Irregular distribution of chromosomes, 303–307, 309, 310
Lamarckiana, 60, 293–295, 307, 308, 310
Swedish, 293
Triploid derived from, 294
Lamarckiana excelsa, 308
Lamarckiana gigas, 293, 302, 310, 312
lata, 293, 307, 308, 310
lorea, 312
nutans, 294
ochracea, 312
odorata, 312
Origin of constant species-hybrids, 295, 311
Origin of tetraploid, 294, 296
Origin of tetraploid from diploid, 310–313
Origin of tetraploid from triploid, 310
Pedigree of triploid and tetraploid, 301
Pollen fertility, 315
Pollen formation, 294
Pollen sterility, 309, 314
pratincola, 293, 310, 312
pycnocarpa, 294, 308
Ring formation, 53, 61, 63, 65–67
Ring-forming species, 310
rubricalyx, 307
semigigas, 293, 294, 308, 310
simplex, 293, 310
simplex nanella, 310
Stability of tetraploid, 307
stenomeres, 293, 310, 312
Sterility in seeds, 295, 302
sublinearis, 293
Suppression of divisions of meiosis, 312, 313, 315
Tetraploid
Endo-duplication, 311
Fertility, 312
Hypothesis of chromosome doubling, 311
OFFERMAN, C. A., 261, 269
OGURA, S., 190
OKABE, S., 309
Oklahoma, University of, 129
OLBRYCHT, T. M., 175, 255, 256, 259
OLIVER, C. P., 15
Orthoptera, 121, 127
Chiasma formation, 122

Paeonia, 122, 127
PAINTER, T. S., 26, 34, 179, 180, 186, 187, 288, 388
Palermo Botanic Garden, 293, 312
Papaver, 313
Paramecia, 47
Paratettix texanus, 159–161, 164–166, 169, 170
PARLOUR, W., 446, 468
Parthenocarpy, 107, 108
Pathology, 27, 29
PATTERSON, J. T., 32, 33, 37, 41–45, 47, 114, 115, 286, 288, 289, 409, 410, 517, 519
PEARL, R., 103, 444
PEARSON, K., 433, 435
PEASE, M. S., 75
PELLEW, C., 61, 311
PERMAR, D., 335, 338, 339, 340, 343, 344, 350, 351, 367, 370, 383
Petri dish, 1, 295
Petrolatum, 194
PETROV, S. G., 68, 77–80, 82, 83, 91, 92
Petunia, 108, 307, 309
violacea, 107
Philippines, 455
Phrynotettix, 406
Pigeon, crossing over in, 92
Pisum, 53, 61, 62, 66
PITT, F., 447, 448, 460–462, 470, 476
PLATE, L., 338
Platypoecilus maculatus, 522, 526, 531
Abnormal distribution of sex factors, 534
Crossing over, 531, 533, 534
Character
Gold, 522–526, 528–530, 533
Description of, 522
Hereditary behavior, 522, 526
Nigra, 523, 526, 528–534
Pulchra, 523, 526, 528–532, 534
Rubra, 523, 525, 526, 528–533
Wild-type, 523–525, 528–530, 532–534
Fertilization, 533
Gene
Blue, 522
Non-red, 523, 525, 526, 532
Non-spotted, 525, 526, 531–533
Non-stippled, 523
Orange, 522
Red, 522, 524–526, 531–533
Silver, 522
Spotted, 522, 523, 525, 526, 531, 532, 534
Stipples, 522–524, 532

Platypoecilus maculatus (Continued)
Gene (Continued)
White, 523, 532, 533
Wild-type, 532, 533
Yellow, 522
Non-disjunction, **531**
PLOUGH, H. H., 269
Poeciliinae, **522, 531**
Bionomic studies on certain teleosts, **522, 531**
Point-mutations, 177
Pollen fertility, 60, 63
Pollen sterility, 53, 57–61, 63, 66
Pollination, 95, 97, 99, 100, 103
Polyploidy, 128
POUND, E. O., 448, 461, 469
Primrose, 133
Primula
Kewensis, 311
sinensis, 309
PRINCE, S. F., 159, 162
Protein, 130–133
Protein molecule, 29, 30
Prunus, 309, 313
PUNNETT, R. C., 72, 75, 86, 91, 210, 212
Pyrus, 309

Rabbit, 233, 238, 242–244
Growth, 221, 222, 246, 248
Radiotherapy, 40
RANDOLPH, 417
RANDOLPH, L., 308
Rat, crossing over in, 93
REDFIELD, H., 535, 545, 547–549, 551, 553–555
REEVES, R. G., 329
REGAN, 248
RENNER, O., 294
RESSOVSKY (See TIMOFÉEFF-RESSOVSKY)
RHOADES, M. M., **535**, 552
Rhoeo, 53, 59–62, 66, 406, 408
discolor, 53, 59, 390, 391
Pollen sterility, 59
RICHARDS, M. H., **129**
RIDDELL, W. H., 450
Ridgway Plate 1, 522
Ridgway Plate 7, 522
Ridgway Plate 17, 148
Ridgway Plate 30, 522
RINKEL, H. J., 131
RITTERHAUS, T., 68, 210, 215, 216, 218
ROBERTSON, D. W., **148**–150, 153, 154
ROBERTSON, T. B., 103
ROBERTSON, W. R. B., 121, 122, 169, 170, 390
Rockefeller Foundation, 193
Rockefeller Institute for Medical Research, **415**
Rosa
relicta, 309
Wilsoni, 311
Roscoe B. Jackson Memorial Laboratory, **481**
Royal Jersey Agricultural Society, 415
RUCKER, E. H., 213
Russian thistle, 133

SABROSKY, C., 161
Salix
caprea, 106
Fusion of homoeotypic spindles, 313
viminalis, 106
Fusion of homoeotypic spindles, 313
SANDERS, A. H., 457
SANSOME, E. R., 61
SANSOME, F. W., 206, 311
SARAO, F. B., 455
SAUNDERS, E. R., 75
SAX, H. J., **121**–123, 127, 128
SAX, K., **53**, 59, 61, 63, 121, 389
Saxifraga Potternensis, 311
SCHMIDT, 324
SCHNEIDER, M., 448, 449
SCHOUTEN, A. R., 293
SCHULTZ, J., 115, 173, 178, 269, 270, 278, **284**, 285
SCHWEMMLE, J., 312
Scilla, 391
sibirica, 390
Segmental interchange, **53**, 60, 67
SEKIGUTI, K., 43
SEREBROVSKY, A. S., 68, 72, 77–80, 82, 83, 91, 92, 210, 211, 214, 215, 217, 218
Sex, influence on crossing over, **92**
Sex ratio, 3, 8, 12–14
Sexual dimorphism, 70
SHARP, L. W., 390
SHEARER, P. S., 452
SHIWAGO, P. J., 68
SHOEBROOK, J., 459
Silkworm, 115
Crossing over, 92, 93
SIMONET, M., 324
SIPE, G. R., 82, 87, 92
Sixth International Congress of Genetics, 193, 293
SMITH, A. D. B., 452, 454
SMITH, J., 129
Solanum, 307–309
Decapitation of stems, 311
lutum, 207
nigrum, 207
STADLER, L. J., 23
Stain
Aceto-carmine, 53, 54, 408, 493
Aceto-haematoxylin, 53
Crystal violet, 493
Crystal violet iodine, 123
Flemming's triple stain, 123
Gentian violet, 372
Gram method, 296
Haidenhaim's haematoxylin, 97
Hyrax, 391, 410
Iron-aceto-carmine, 390
Iron alum-haematoxylin, 199, 296, 493
Iron-brazilin, 390–392, 408
Iron-haematoxylin, 7, 408
STEERE, W. C., 309, 493
STEIN, E., 487, 493, 518, 519
Stenobothrus, 407
Sterility, 56, 57, 108, 114
STERN, C., **26**, 43, 46, 174, 189, 190, 258, 388, 536
STEVENS, M. M., 68
STOMPS, T. J., 293, 294, 307, 310, 312
STONE, W. S., 269
STOTTLEMEYER, 248
STRASSBURGER, 294
STROMAN, G. N., 329
STUDY, 129, 131, 146

STURTEVANT, A. H., 16, 18, 19, 21–23, 25, 53, 114, 115, 173, 174, 176, 179, 190, 261, 271, 274, 278, 388, 402, 535
Sulphydryl, 243
SUMNER, F. B., 222, 244
Suppression of heterotypic division, 312, 315
Suppression of homoeotypic division, 312, 313
SUTTLE, A. D., 82, 87, 92
SUTTON, W. S., 390
Sweet pea, 218
SWETT, W. W., 222, 223, 226, 229, 238, 247

TAKAHASHI, W., 493
Tallquist color scale, 373
Tallquist method in hemoglobin determinations, 379
TANAKA, Y., 115
Telmatetlix aztecus, 159
TEMPLETON, G. S., 472
TERENTIEVA, E. L., **117**
Terminalization, 65
Tettigidea parvipennis pennata, 159
Texas Agricultural Experiment Station, **329**, 330
Texas, University of, **32**
THADANI, K. I., 330
THOMPSON, W. P., 313
Thymonucleic acid, 30
TIMOFÉEFF-RESSOVSKY, H. A., 289
Timothy, 133
TINIAKOW, G. G., **117**, 119
Tomato, **193**, 199, 205, 208, 218
  Callus tissue, 193
  Chromosome size, 208
  Cross-sterility, 194, 208
  Cytogenetic studies, **193**
  Domesticated species, 207
  Fertility, 194–197, 206, 208
  Fruit, placental tissue of, 194
  Genetic observations, **206**
  Haploid, **194**, **200**–202, 204, 205, 207, 208
  Micromorphological observations, **203**
  Pollen abortion, 193, 195, 197, 198
  Pollen sterility, 198, 203
  Self-fertilization, 194, 208
  Sterility, 193–198, 206, 208
  Tetraploid, **193**, **194**, **195**, **196**–198, **200**, 201, **202**–209
    Anaphase, **202**, 208
    Artificial production, 193, 194
    Breeding behavior, 208
    Chromosome pairing, 207–209
    Conjugation, 207
    Cytological behavior, 204
    Cytological observations, **198**
    Decapitation-callus method, 194, 197, 201, 205, 208
    Diplotene, 199, 200, 202, 204
    Disjuction, **202**
    From F_1 of species cross, **196**
    Gene
      Compound-cluster, 195
      Dwarf, 193, 195
      Ovate, 195
      Peach, 195
      Potato-leaf, 193
    Metaphase, 199, 201–205, 208
Tomato (Continued)
  Tetraploid (Continued)
    Micromorphological observations, **203**, 205
    Pachytene, 200
    Parasynaptic pairing, 200
    Pollen development, 208
    Pollen diameter, 205
    Seed development, 208
    Segregation, 206, 209
    Species cross, **202**, 204–206
      Genetic behavior, 206
    Species hybrid, 203, 204, 207
    Synapsis, 200
    Variety
      Marglobe, 195
      Model, 193, 197
    Wild species, 207
Toronto, University of, **210**
Tradescantia, **53**–55, 59, 61–67, 406, 408
  bracteata, 53–55, 59, 64
  Chiasma frequency, 63
  Chromosome pairing, **53**
  edwardsiana, 53–59, 61–64, 66
  Fertility, 56, 57, 66
  Fertilization, 46, 49
  fluminensis, 391
  gigantea, 53, 55
  occidentalis, 53, 55, 62
  pilosa, 55
  reflexa, 53, 55, 58, 59, 62, 63, 66
  Segmental interchange, 62–64, 66
  virginiana, 390, 391
Translocation, 261, 262, 286, 287, 289
Trenner pipette, 372
Triticum, 313
TROWBRIDGE, P. F., 226, 232, 237
Tsuga, **121**, **123**, 125, 128
  canadensis, 123, 124
  Chiasma formation, **121**–123, 125–128
  Chiasmata and chromatids, **123**
  Diakinesis, 123, 125
  Terminalization, 123, 128
Tulipa, 307, 309, 391
TURRILL, W. B., 311
Twins, identical, 144, 145

ULLMAN, 131
Ultra-violet light, 29
UNGER, 130, 131
University
  California, **221**, 224, 230, 240, **388**, **487**, 493, **522**, **531**
  Chicago, 522
  Cornell, **535**
  Harvard, 53, **95**, **324**, 335, 338
  Johns Hopkins, 415
  Michigan, 58, **293**
    Botanical Garden, 58
  Minnesota, **82**
  Moscow State, Zoological Institute, **117**
  Oklahoma, **129**
  Texas, **32**
  Toronto, **210**
Urtica gracilis, 391
Uvularia, 407

VAN OVEREM, 308
VERHULST, P. F., 103
Vertebrates, 218
VILMORIN, DE, R., 324
Vitamin A, 71
VRIES, DE, H., 293, 294, 310, 312

WAGNON, K., 248
WALLACE, R., 457, 472
WARE, J. O., 330
WARREN, D. C., **68**, 82, 83, 91, 210, 211, 215–218
WATSON, J. A. S., **447**
WEBB, H. F., 448, 449
WEBBER, J. M., 493
WEBER, A. D., 441, 472
WEIER, E., 296
WEINSTEIN, A., 275
WENRICH, D. H., 121–123, 127, 390, 406, 410
WENTWORTH, E. N., 441, 444, 445, 448, 456, **472**, 479
WETTSTEIN, VON, F., 536
WHITING, P. W., 535
WIETZ, W., 130
Will certified pipette, 372
WILSON, I., 133
WILSON, J., 457
WINCHESTER, A. M., 37
WINGE, Ö., 295, 311
WINKLER, H., 193, 311
WRIEDT, C., 92, 446, 447, 457, 465–467
WRIGHT, S., 221, 222, 238, 244, 246, 433, 447, **484**
WYCKOFF, R. W. G., 1

X-ray, 1–6, 10–13, 15–18, 21, 22, 25, 28, 29, 32, 33, 35–40, 42–45, 48, 50, 51, 63, 173, 174, 180–182, 189, 191
Xanthophyll, 71, 329, 332

Yak, 465
YASUI, K., 312
Yeast, 30

Zea, 53, 60, 61, 63, 66, 179, 307–309, 408, 409
  Crossing over, 535, 536, 542, 546
  Crossing over in triploids, **545**
  Double strand crossing over, **536**, 544, 554
  Fertilization, 546
  Floury-flinty endosperm, 538
  Gene
    Green seedling, 537, 539
    Purple, 537, 538, 540, 542, 543
    Red, 537, 538
    Virescent seedling, 537
  Origin of a 32 chromosome plant, **543**
  Self-pollination, 542
  Trisomic ratios, **544**
Zebu, 465
Zeiss apochromatic, 392
ZELENY, C., 23
ZIRKLE, C., 98
Zoological Institute of Moscow State University, **117**

C 30